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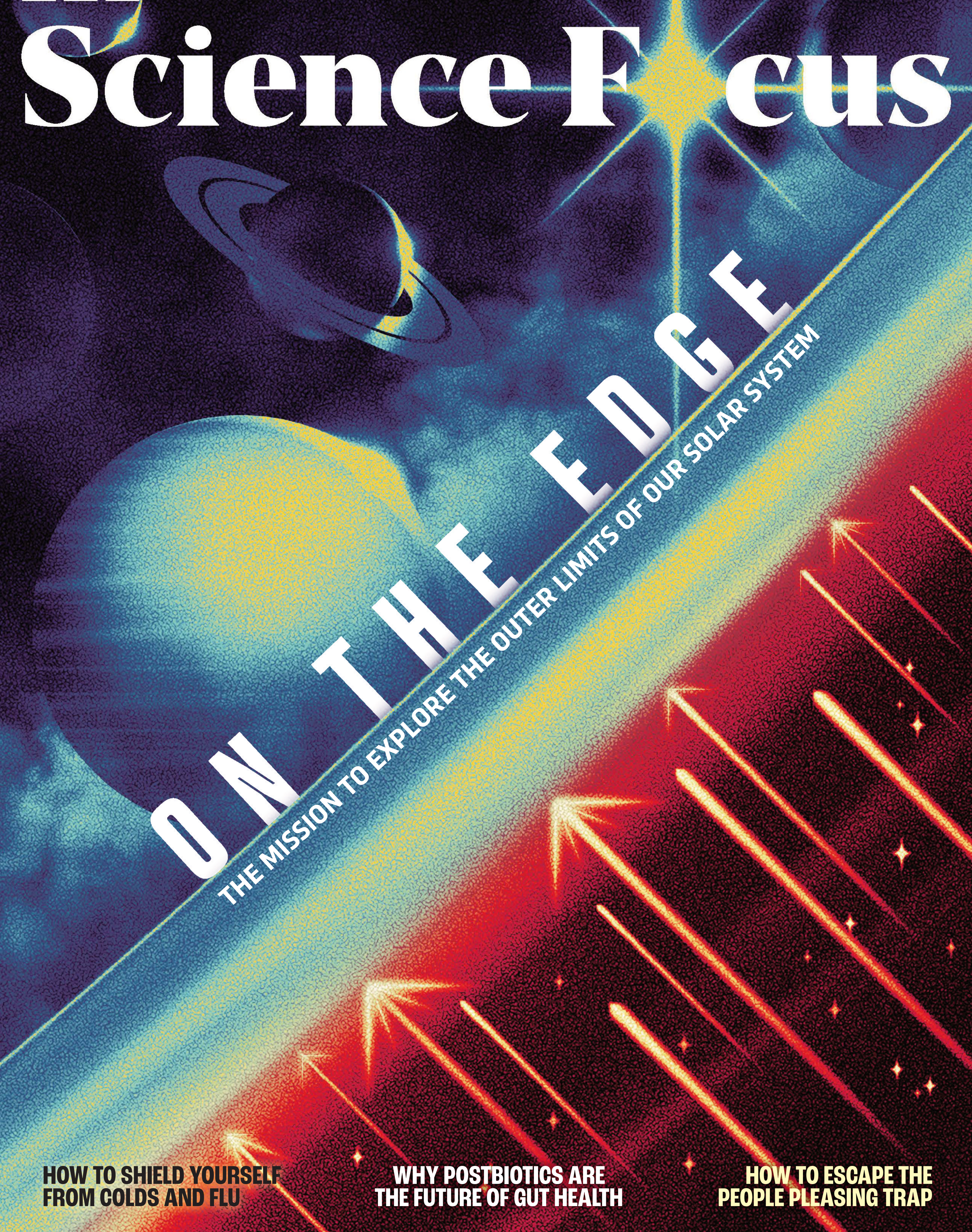
THE MYTH OF HIGH-CHOLESTEROL FOODS, DEBUNKED

Science Focus



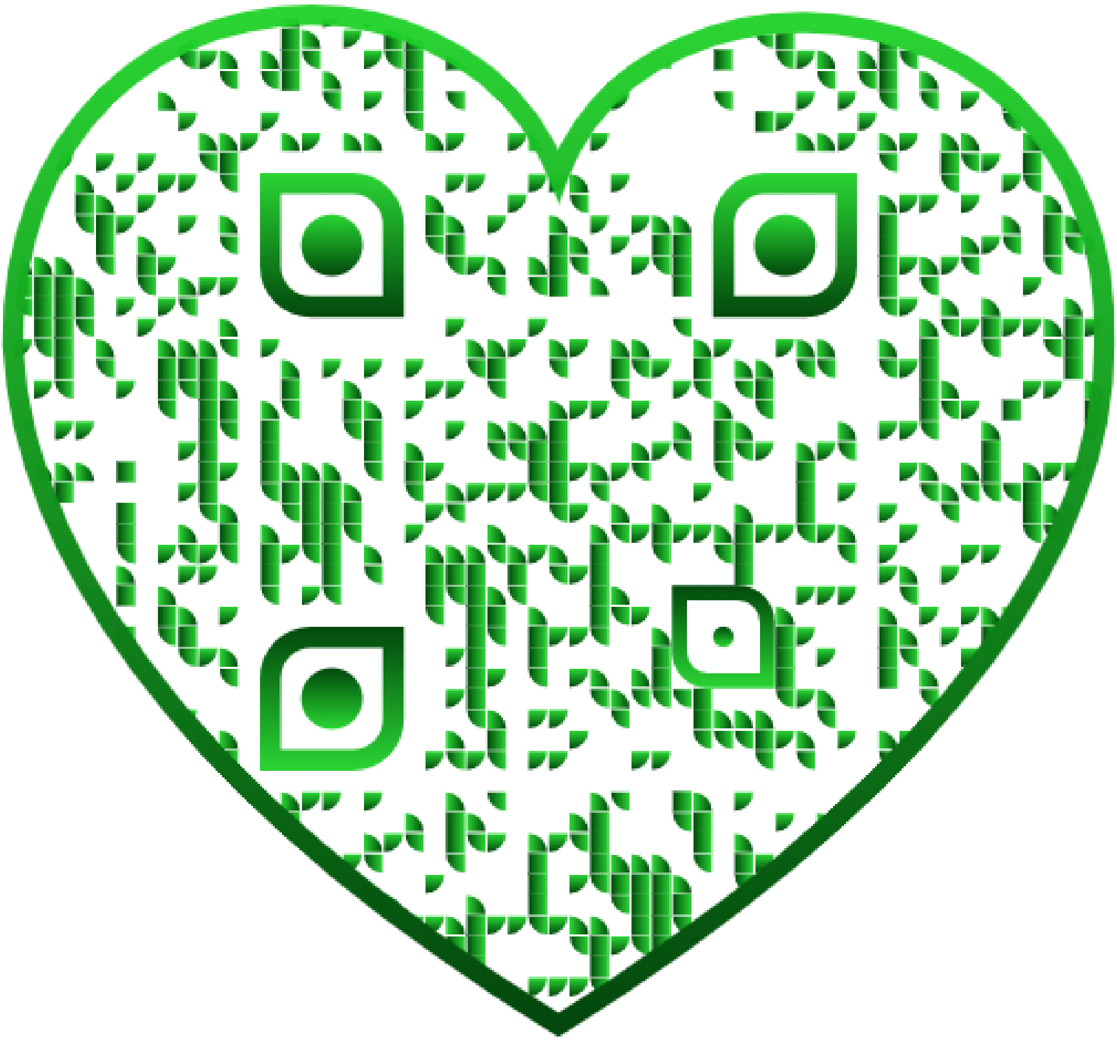
ON THE EDGE

THE MISSION TO EXPLORE THE OUTER LIMITS OF OUR SOLAR SYSTEM

HOW TO SHIELD YOURSELF FROM COLDS AND FLU

WHY POSTBIOTICS ARE THE FUTURE OF GUT HEALTH

HOW TO ESCAPE THE PEOPLE PLEASING TRAP



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FROM THE EDITOR

I wonder how many discoveries in human history were made because someone thought: “Let’s take a look around the corner”. This time, the corner – figuratively speaking – is the region of space beyond Pluto, but still within what we consider the edge of our Solar System. More precisely, I’m talking about the area where the Sun’s influence begins to fade – the boundary of the heliosphere.

Think of the heliosphere as a vast bubble, emanating from the Sun, that envelops our Solar System. Solar winds – streams of energised particles – blast out from our star in all directions, but eventually they fizzle out. Where the winds are strong, they push back more harmful cosmic radiation gusting in from elsewhere in our Galaxy, shielding us. But the further these winds travel, the weaker they become, until, eventually, the solar particles become inconsequential. This is the place that scientists consider to be the edge of our Solar System and the beginning of the interstellar medium – the space between the stars.

Here, space roils with a cosmic zoo of exotic particles from strange places. This is exactly what NASA’s recently launched Interstellar Mapping and Acceleration Probe (IMAP) wants to study. Its mission is to make sense of the stuff that’s arriving here from other parts of space, to understand how our Sun forms a barrier that protects us from the more harmful elements out there, and to chart what’s going on at the very edge of what we know. Get the full story on p60.

Daniel Bennett

Daniel Bennett, Editor

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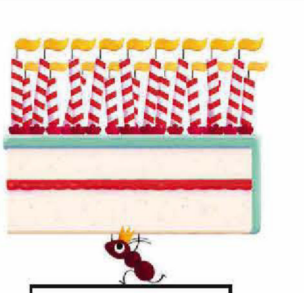

CONTRIBUTORS

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NASA’s newly launched probe will study the edge of our Solar System. Science writer Jonathan reveals what it hopes to find. p60

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Jane Goodall: Beauty and the Beasts
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Watch now on BBC iPlayer

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This four-part series follows wildlife cameraman Hamza Yassin as he uncovers the best of the British countryside through each season.
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The Evidence: Weight loss jabs
Are weight loss injections the new ‘wonder drug’? Host Claudia Hammond asks a panel of experts about what the latest studies on medications such as Ozempic and Wegovy are revealing.
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CONTACT US

Advertising
David.DSouza@ourmedia.co.uk
0117 300 8110

Letters for publication
reply@sciencefocus.com

Editorial enquiries
editorialenquiries@sciencefocus.com
0117 300 8755

Subscriptions
ourmediashop.com/contact-us
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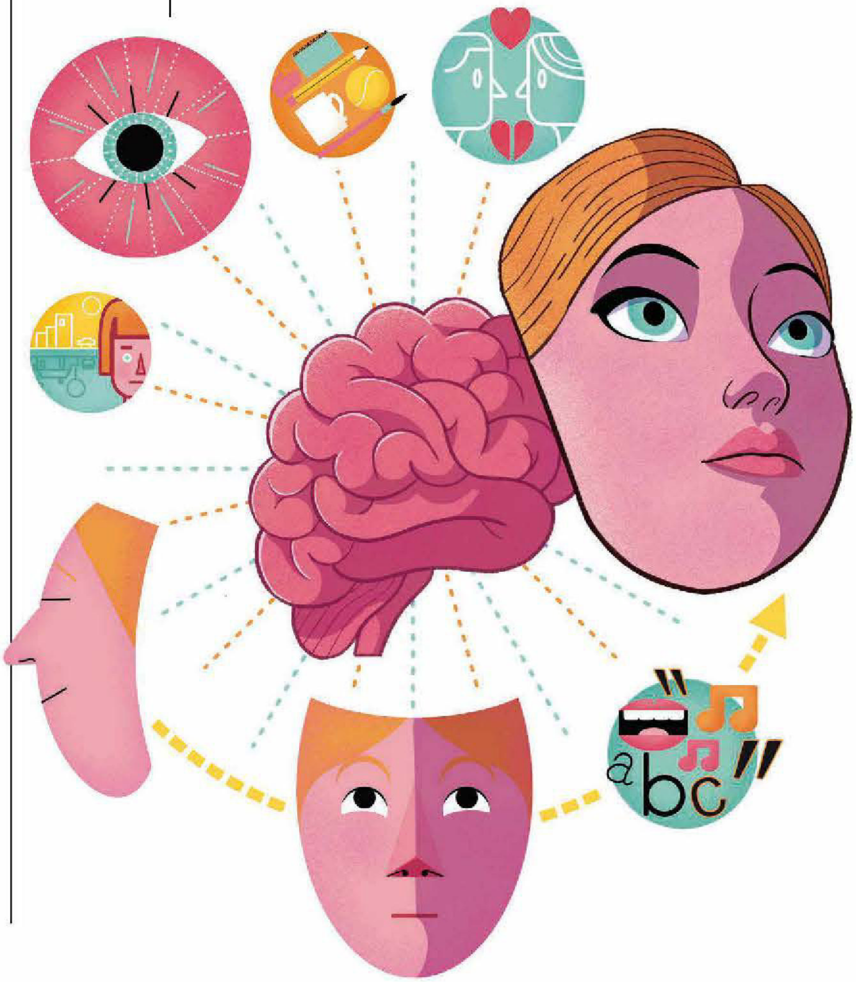
How does my brain create my sense of self?



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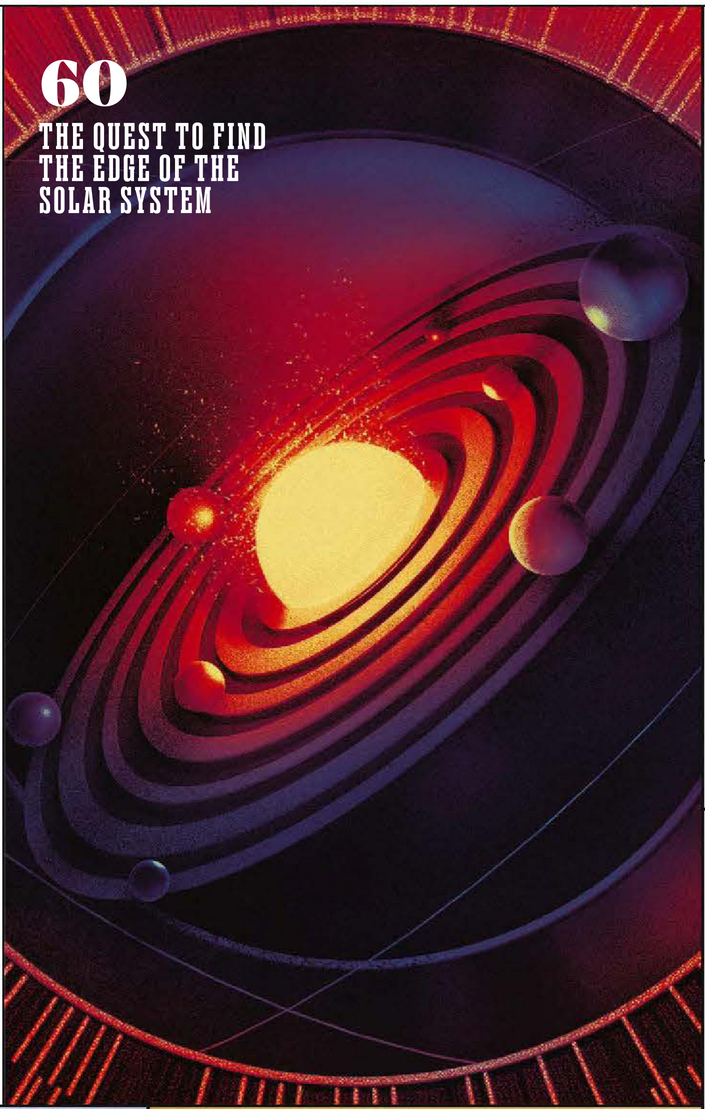

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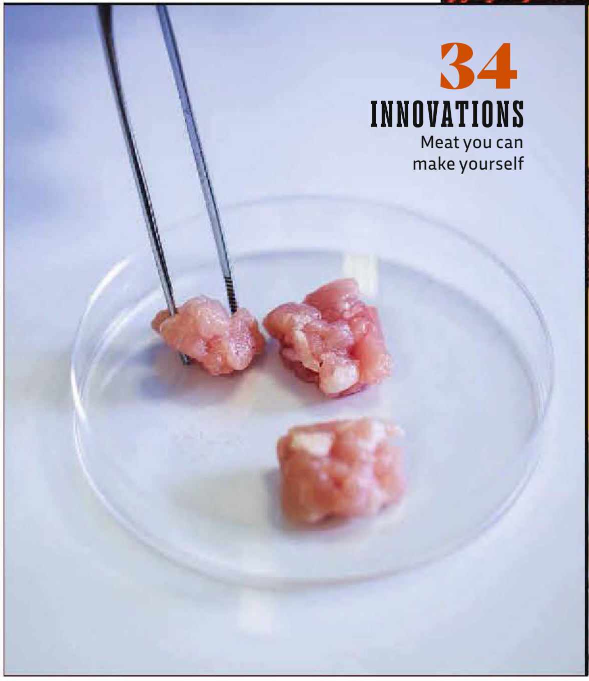

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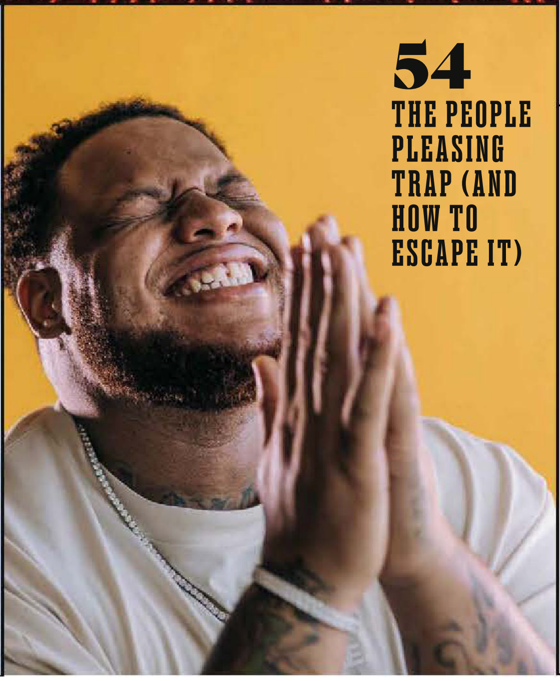

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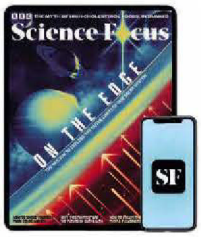

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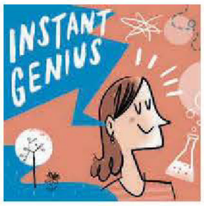

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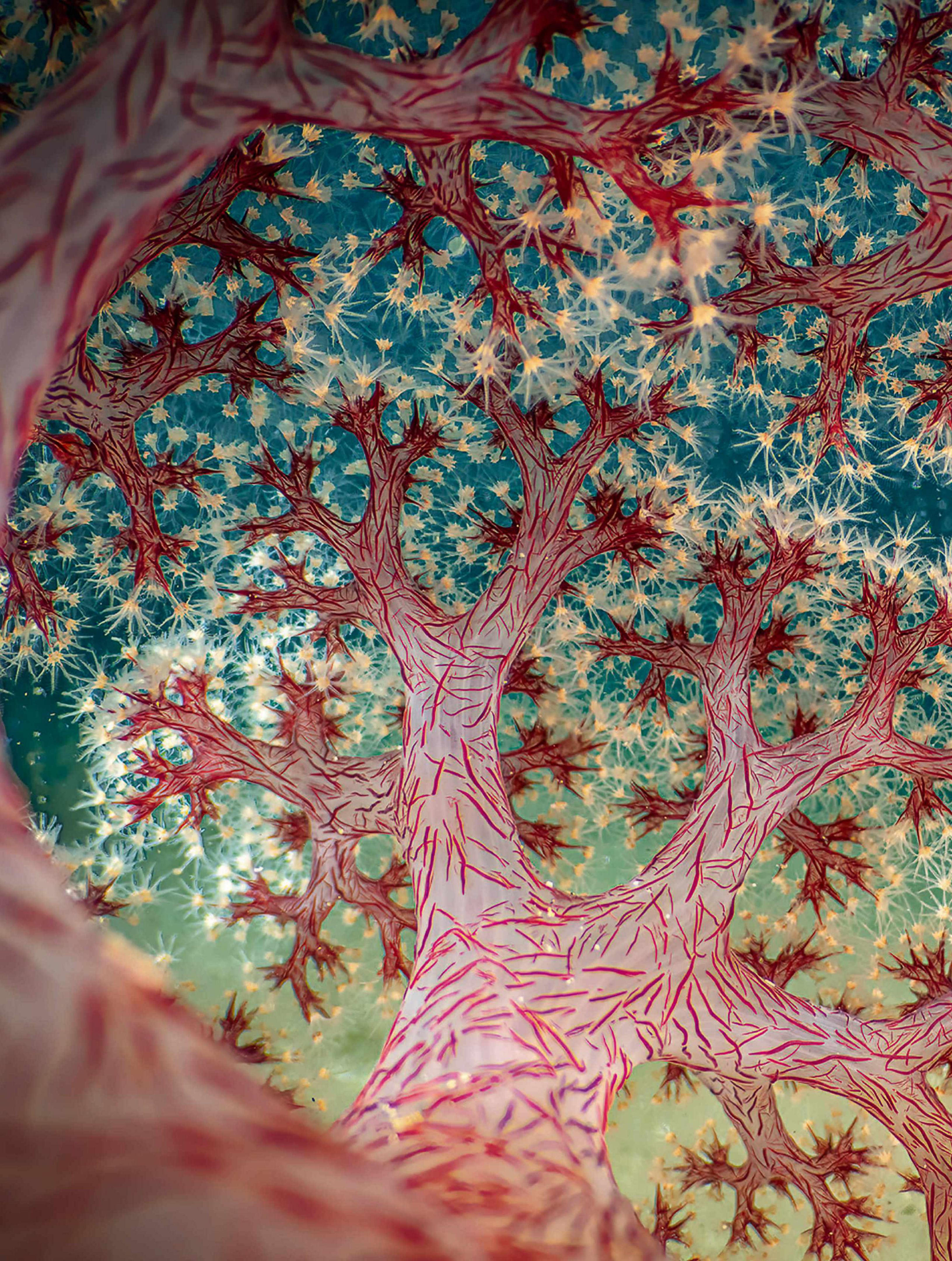

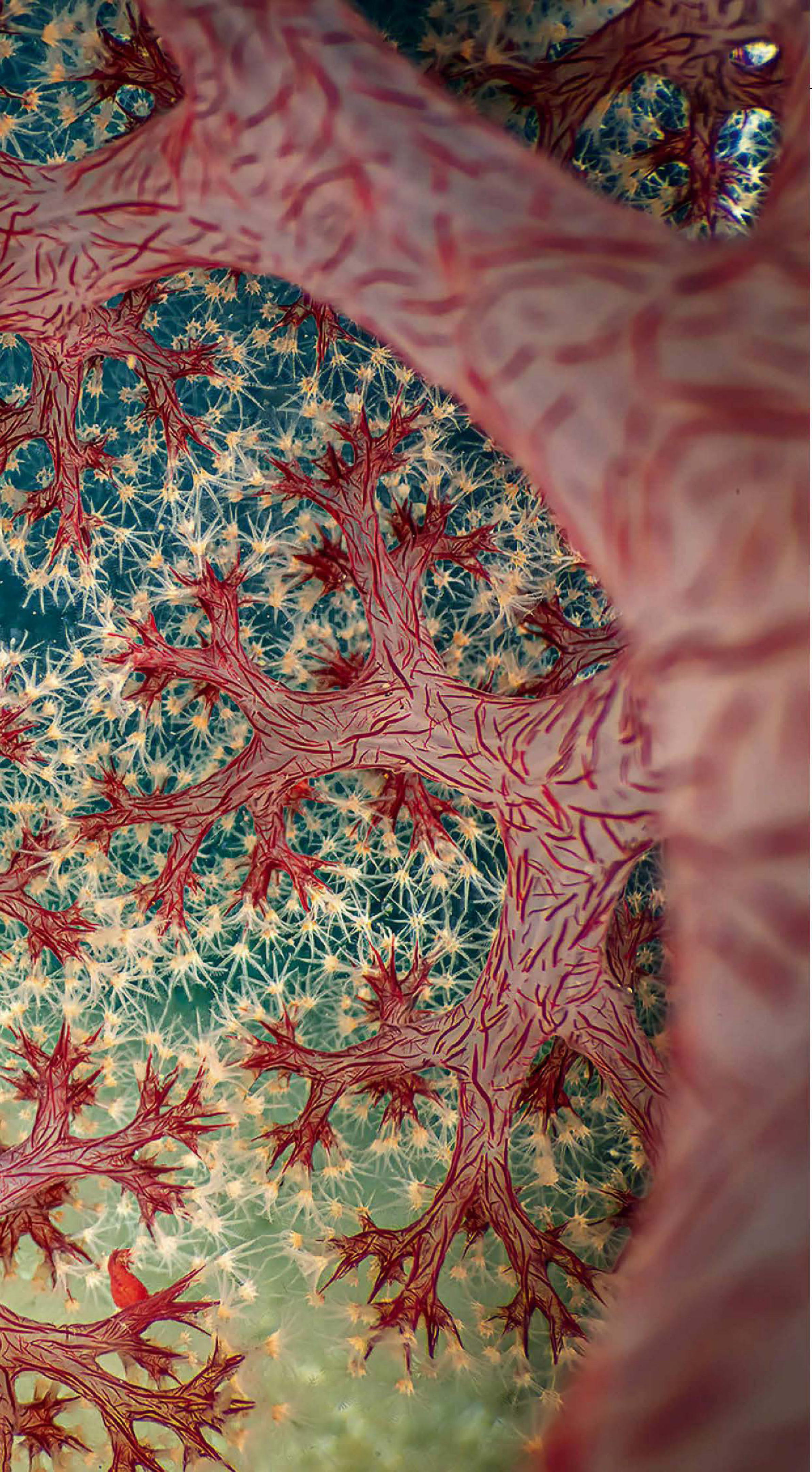

EYE OPENER

Can't see the coral for the trees

LEMBEH STRAIT, **INDONESIA**

This may look like the sort of fantasy forest you might get lost in during a dream, but it's very much real. That said, it's neither a forest, nor big enough for you to explore. And that's because the surreal structures you see here aren't trees. They're coral. Specifically, a cauliflower soft coral, but seen from the inside.

The image, which was a runner-up in the Natural Artistry category of the 2025 Wildlife Photographer of the Year Competition, was taken by Ross Gudgeon. He captured it with the help of an extended macro wide camera lens (a specialised system that helps make very small things look very large), which he managed to carefully thread up inside the coral's delicate branches.

Most corals rely on photosynthesis for sustenance, but soft corals, like this one, are filter feeders. Each of the branches you see in this image is tipped with a polyp – essentially a tube with a mouth on the end. Around that mouth are feathery tentacles that comb the water currents, hoping to catch any passing phytoplankton and pass it back into the polyp's hungry opening.

ROSS GUDGEON / WILDLIFE PHOTOGRAPHER OF THE YEAR

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EYE OPENER

Surf's up

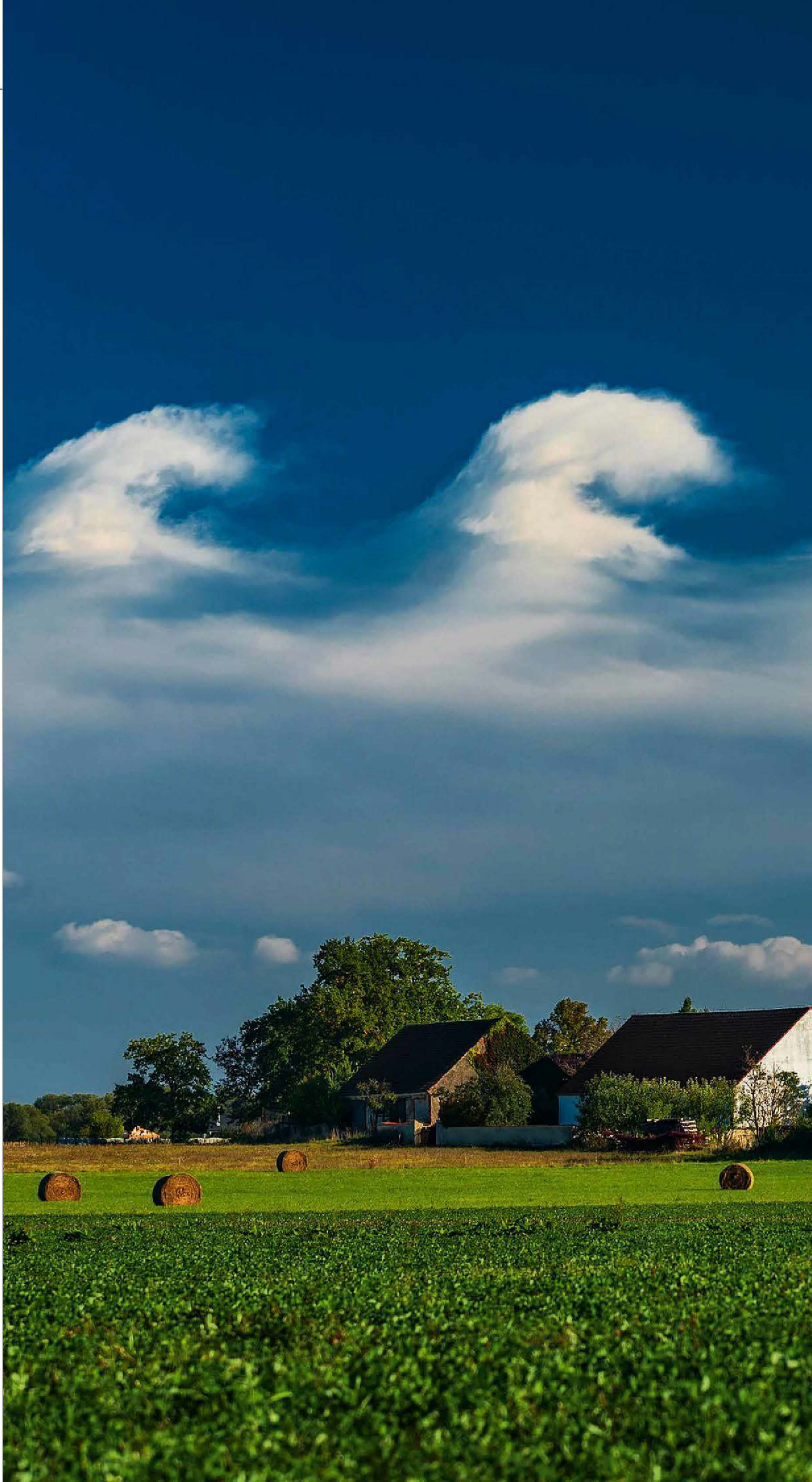
VODŇANY, **CZECH REPUBLIC**

These distinctive clouds are named after the two physicists – William Thomson (Lord Kelvin) and Hermann von Helmholtz – who studied the phenomenon that forms them. The peculiar wave-like shape of Kelvin-Helmholtz clouds is caused by two layers of the atmosphere moving at different speeds above and below the cloud layer.

When warmer, less dense air above the cloud is moving faster than the cooler, denser air below, it causes ripples to form on the top of the cloud, much like how wind causes ripples on a lake. The warmer, faster air scoops up the water vapour that forms the cloud and carries it along for a while. A few moments later, gravity gets the better of the warm air and brings the vapour back down, like a wave crashing on the shore.

These formations can occur anywhere in the troposphere – the lowest part of Earth's atmosphere – but only hold their shape for a few minutes. Hence, spotting and photographing them is a rare occurrence.

Kelvin-Helmholtz clouds are also considered to be a tell-tale sign of turbulence. If airline pilots spot them, they're encouraged to steer clear instead of attempting to ride the waves.



LUKÁŠ GALLO/ STANDARD CHARTERED WEATHER PHOTOGRAPHER OF THE YEAR

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EYE OPENER

Egg timer

SHIYAN, **CHINA**

This is a dinosaur egg. But it's also a key, because it has helped the experts examining it to unlock a new, more accurate method of dating dinosaur eggs.

Up until now, the ages of fossilised eggs were estimated based on the analysis of radioactive elements in any volcanic rocks surrounding them. That method wouldn't work here, though, as there are no volcanic rocks near where this egg was found. There is, however, uranium in the crystals that formed inside the egg over the millennia it lay buried – an inclusion that opened the door to another technique: uranium-lead dating.

The technique uses a mass spectrometer to determine the ratio of uranium to lead in a material and thereby pinpoint its age (radioactive uranium decays through a series of steps into stable lead isotopes over a known timescale). It's been used to date rock, teeth and bones before, but never a fossilised egg.

It worked well on the crystals, but, to the experts' surprise, it worked even better on the eggshell itself. Based on their results, the experts believe the egg was laid around 85.91 million years ago. Prior to this analysis, their best guess at dating the specimen was sometime during the Late Cretaceous period, from around 100–66 million years ago.

DR. BI ZHAO

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Another dull watch from Christopher Ward

CHRONOMETER
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Christopher
Ward

christopherward.com

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LETTER OF THE MONTH

Faulty plan

The fascinating article on 'Heatquakes' by Prof Bill McGuire (September, p54) – exploring how higher rainfall could lead to more earthquakes – got me thinking about some research I'd seen before.

Geologists were considering whether to inject tectonic faults with water in order to lubricate them. This would result in a larger number of small earthquakes, instead of letting tension build up until a catastrophic quake happens. Could this method help?

Alan Blackwood, via email

Author Prof Bill McGuire replies…

We'd have no control over the outcomes of the water-injection method, especially if we don't know how much strain has already built up around the plates. Rather than promoting smaller quakes, there's a good chance the injections could trigger the main event.

Unfortunately, the faults most likely to rupture would be those that have accumulated the most strain, which are the ones most likely to trigger major, destructive earthquakes.

WRITE IN!
The writer of next month's Letter of the Month will receive two popular science books: ***The Secret Life of the Universe*** and ***Horses***, worth over £30.

WORTH OVER £30

THE SECRET LIFE OF THE UNIVERSE
LUDOVIC ORLANDO
HORSES
A 4,000-YEAR GENETIC JOURNEY ACROSS THE WORLD

ALAMY X2

Mind over media

As a 71-year-old woman with an interest in "mind over matter" topics, I found David Robson's article 'You're only as old as you think' (September, p62) interesting.

What it didn't mention, however, is the huge role the media plays in promoting negative stereotypes about ageing. Media influence on our attitudes is immense and as such, can't be ignored.

Every day, advertisers tell us that we should colour our hair, "correct" our wrinkles and remain youthful at all costs. In the media, being "old" is associated with being forgetful, clumsy and unable to keep up with life.

The fulfilment people seek in old age may well involve letting go of the endless quest for youth. Instead, we should find and nurture what makes us happy as individuals.

Elena Holden, via email

Pause for thought

I read Dr Sam Carr's recent Reality Check article (September, p28) on the unintended consequences of the UK's age verification law for accessing pornography.

What struck me most was that even a small delay, such as the time taken to verify age, can interrupt an impulsive urge. This reminded me of suicide-prevention strategies, where delaying access to lethal means, has been shown to save lives.

In both cases, the psychology is the same: impulses often lose their power when they are met with resistance. Age verification laws may therefore provide a vital pause that helps to shift behaviour.

Kattamreddy A R, Nellore, India

Adult content 18+
confirm your age
I am 18 years old or older
I am under 18 years old

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CONTRIBUTORS
Amy Arthur, Claire Asher, Craig Beall, Alicia Blasco, Robin Boyden, Dean Burnett, Sandrine Ceurstemonte, Sam Freeman, Adam Gale, Daniel Garcia, Alastair Gunn, Christian Jarrett, Pete Lawrence, Nish Manek, Holly McHugh, Jonathan O'Callahan, Helen Pilcher, Ana Santi, Kyle Smart, Tim Spector, Ian Taylor, Magic Torch, Luis Villazon, Quentin Willson and all the folks at VMG.

ADVERTISING & MARKETING
Business development manager David D'Souza
Newstrade manager John Lawton
Head of acquisitions Clive Evans-Morris
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TECHNOLOGY
Senior marketing manager – digital editions Kevin Slaughter
Tech director Azir Razzak

INSERTS
Laurence Robertson 00353 876 902208

LICENSING & SYNDICATION
Director of international and licensing Tim Hudson
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CONTENT OPERATIONS
Content operations, procurement & sustainability director Sarah Powell
Group content operations & sustainability manager Louisa Moulter
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CEO Andy Marshall
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BBC STUDIOS, UK PUBLISHING
SVP licensing Stephen Davies
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UK.Publishing@bbc.com
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EDITORIAL COMPLAINTS
editorialcomplaints@ourmedia.co.uk

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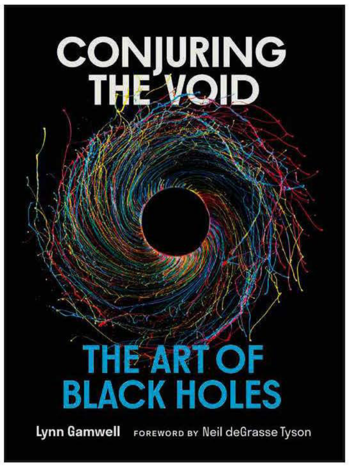

CONJURING THE VOID
THE ART OF BLACK HOLES
Lynn Gamwell
FOREWORD BY Neil deGrasse Tyson

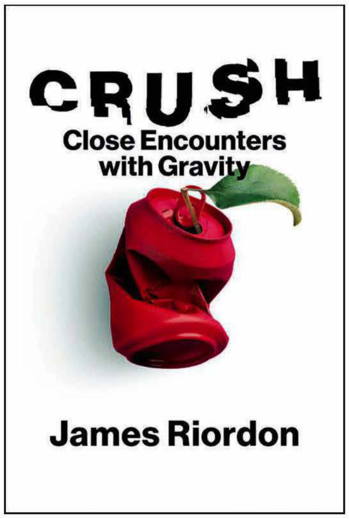
Conjuring the Void

The Art of Black Holes

By Lynn Gamwell

“A visually arresting voyage through a fascinating convergence of art and science...”

—Brian Greene, author of *The Elegant Universe*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Close Encounters with Gravity
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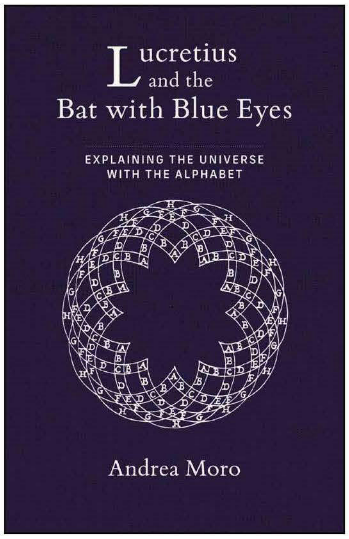
Crush

Close Encounters with Gravity

By James Riordon

“A fascinating exploration.”

—Scientific American


Lucretius and the Bat with Blue Eyes
EXPLAINING THE UNIVERSE WITH THE ALPHABET
Andrea Moro

Lucretius and the Bat with Blue Eyes

Explaining the Universe with the Alphabet

By Andrea Moro

“Andrea Moro undertakes an engaging voyage through one of the most fascinating voices of antiquity.”

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Hygiene isn't the only health issue linked to phone use on the toilet **p20**

GANNET PLANET
Astronomers spot a distant planet with an insatiable appetite **p22**

DAMNED IF YOU DO...
How taking vitamin D could lower your vitamin D **p22**

NEWS FROM THE FRONTIERS OF SCIENCE

DISCOVERIES

MEDICINE

'Ageing atlas' reveals how time reshapes our genes

Scientists are building the clearest picture yet of how we age – right down to our cells and DNA

The visible signs of ageing reflect complex genetic processes that scientists are learning more about

The visible signs of ageing – wrinkles, greying hair, aching joints – are only the surface expression of something far more intricate happening inside our cells. Beneath the surface, every organ in our bodies undergoes a subtle molecular transformation as we grow older.

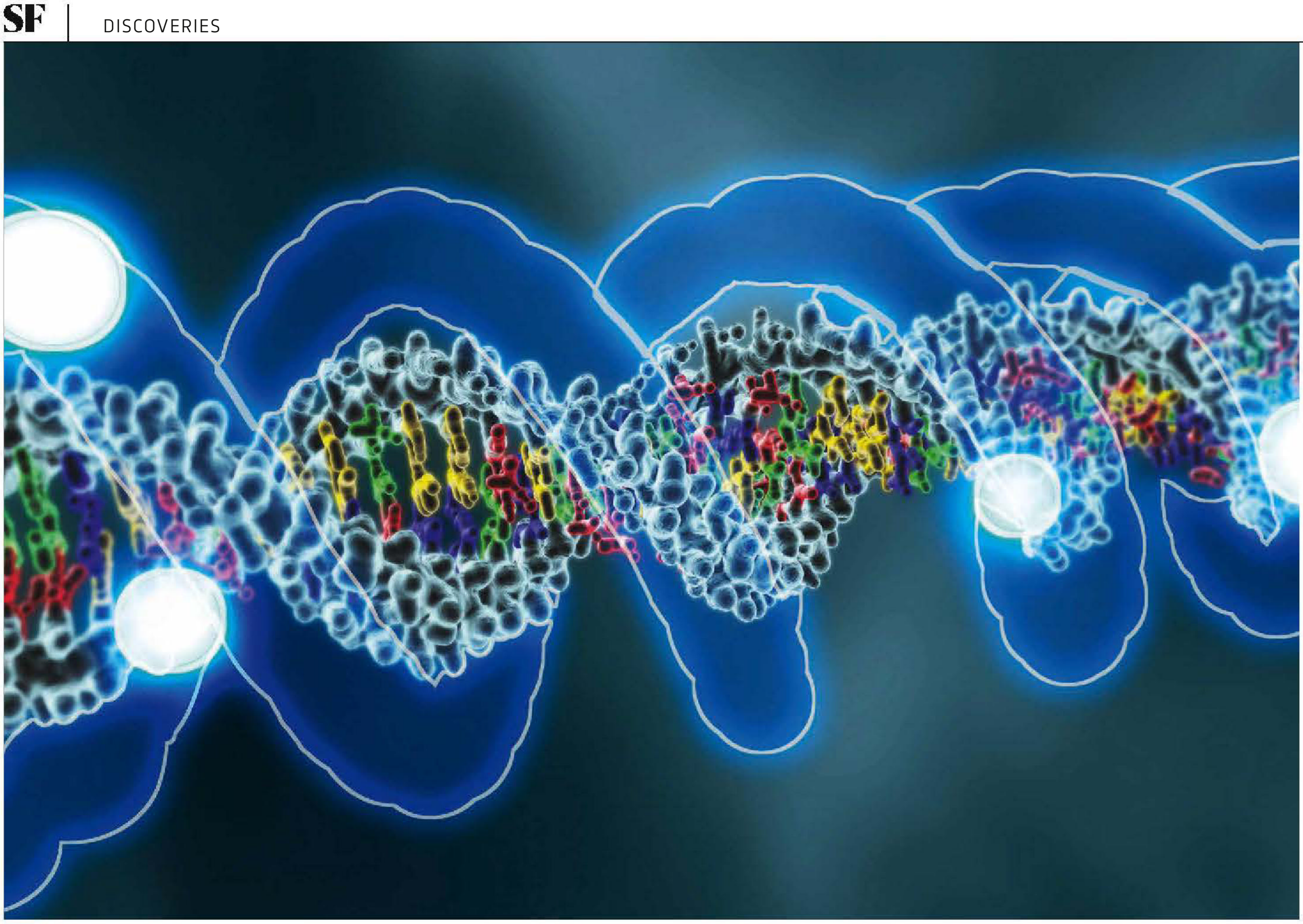
Now, scientists have built the most comprehensive map yet of how that process unfolds.

Drawing on data from more than 15,000 samples, the findings – detailed in a preprint study awaiting peer review – offer an unprecedented view of how ageing rewrites our genome's operating manual from head to toe.

Researchers from around the world teamed up to create a sweeping 'ageing atlas' that charts DNA methylation (chemical tags that regulate gene activity) across 17 types of human tissue, tracking changes as we grow older.

"DNA methylation, very simply, is a chemical modification on your DNA," Dr Jesse Poganik, an instructor in medicine at Harvard Medical School and

GETTY IMAGES



ABOVE The expression of a certain gene is altered when methyl groups (the glowing spheres here) attach to the gene's DNA

one of the authors of the new study, told *BBC Science Focus*. "At the very basic level, the main function is to control which genes get expressed and which don't."

Each of your cells has, essentially, the same genetic information in the form of your genome. Yet somehow, the cells in your lungs behave differently from those in your stomach, skin and brain. How do they know what to become and what to do? This is the role methylation performs.

"Depending on the methylation or unmethylation status of particular points on the genome, the expression of particular genes is turned on or off," Poganik said.

But what does all of this tell us about ageing? DNA methylation is one of the body's key epigenetic mechanisms – molecular switches that turn genes on or off without altering the DNA code itself. By attaching or removing small molecules called methyl groups, cells can fine-tune which genes are active in response to diet, exercise, infection and other environmental factors around us.

As the years pass, these methylation patterns shift in characteristic ways, forming the basis of so-called epigenetic clocks – molecular measures of biological age. Until now, most of those clocks have been based on blood samples, leaving scientists unsure whether other tissues follow the same rules.

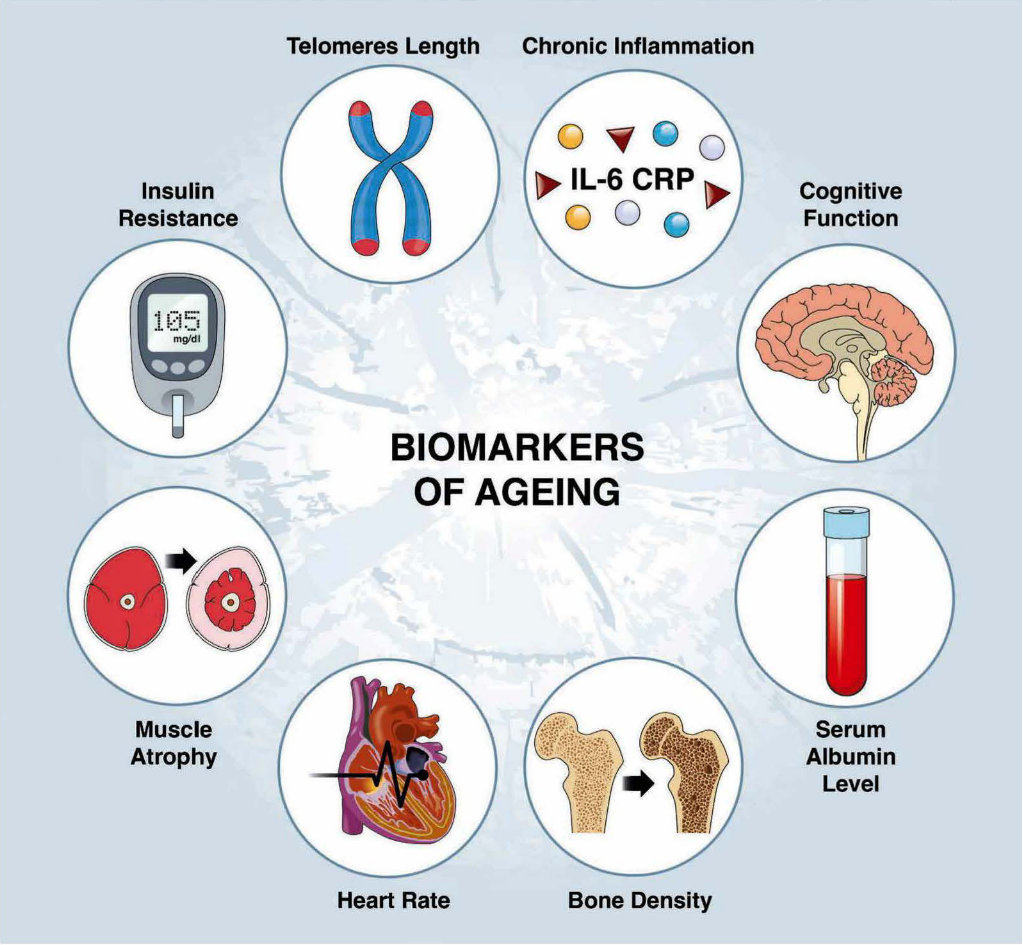
"Those DNA methylation patterns are different between tissues – they're tissue-specific, even cell-type-specific," Prof Nir Eynon, senior author of the study and a research group leader at Monash University, told *BBC Science Focus*. "So measuring blood doesn't necessarily reflect what's happening in the liver, the muscle or the brain."

That difference motivated the researchers to gather every publicly available dataset on methylation they could find, supplemented by new data from international collaborators.

The result: an analysis covering 17 organs, from the brain and heart to the skin, liver, stomach and retina, analysed at nearly a million points across the genome.

> **"Scientists know methylation changes as people age, but they don't know whether those changes cause ageing or whether ageing causes those changes"**

SCIENCE PHOTO LIBRARY, ALAMY ILLUSTRATION: ACUTE GRAPHICS



ABOVE The effects of ageing appear throughout the body, but when and how they show up varies from person to person

AN ATLAS OF AGEING

The researchers discovered the portion of the genome carrying methylation tags varies widely between tissues – from about 38 per cent in the cervix to more than 60 per cent in the retina. Yet, the changes with age were remarkably consistent: most tissues showed a gradual shift towards hypermethylation as we age, where more DNA sites became tagged and certain genes switched off.

Two tissues, however, bucked the trend. Skeletal muscle and lung tissues lost methyl tags over time, potentially leading to overactive or unstable gene expression.

"Most tissues show more hypermethylation with age," Dr Macsue Jacques, the study's first author, told *BBC Science Focus* by email. "But when you break it down to methylation fractions, you actually start to untangle tissue-specific patterns."

For example, fat tissue shifts almost entirely toward hypermethylation, while in the brain, the changes are more balanced. These patterns, Jacques said, may reveal how different organs respond to shared ageing pressures such as inflammation.

Overall, the brain, liver and lung tissues exhibited the largest age-related methylation changes, though the skin and colon also changed significantly. Pancreas, retina and prostate tissues, meanwhile, showed the fewest detectable age-associated changes, possibly reflecting limited data or greater resistance to ageing.

CORRELATION, NOT CAUSATION

At first glance, the data seem to suggest that some organs age faster than others. But the researchers cautioned that these differences can't yet be interpreted as literal rates of ageing.

Part of the reason is statistical: some tissues were represented by thousands of samples, others by only a few dozen. Another part is relational. "We know changes in methylation happen as a function of ageing," Poganik said. "We don't know the extent to which they're causal to the ageing process."

In other words, scientists know methylation changes as people age, but they don't know whether those changes cause ageing or whether ageing causes those changes.

Poganik believes it's likely that the methylation changes are at least partly to blame for some of what we see in ageing. "Even a conservative scientist would tell you there's some element of causality going on," he said.

What makes this new atlas so compelling, he added, is the way it reveals a shared molecular theme running through the body.

"One of the most interesting things about this study is that it shows there's some sort of universality to the ageing process. When we look across the different tissues, we find many of the same methylation changes, and that suggests there's something universal about the process."

Still, he cautioned that not every change is causal. With so many changes in methylation going on, it's almost certain that some are part of ageing, while others are inconsequential.

The ageing atlas may not reveal which changes are important and which aren't, but it does provide a treasure trove of data for scientists to explore that question in more detail.

The atlas is freely available through an online portal for other scientists to use. "We've always focused on open-source research," Jacques said. "And with this, we want to not only advance research by making it available to all, but also initiate collaborative efforts."

Next, the researchers plan to look into some of the universal links they found stretching across all tissues with age, as well as looking at other biomarkers that may be influencing the ageing process.

"The progress that has been made in ageing pales in comparison to the progress that has been made in cancer," Poganik said. This atlas may help scientists close that gap.

ENVIRONMENT

Only 1% of the world is eating a healthy and sustainable diet

A major report found healthier diets could transform the food system

A change in our approach to food production could have positive effects on our health and that of the planet

GETTY IMAGES

> **“If everyone ate a healthy diet, we would be able to feed 10 billion people in 2050 with seven per cent less land than we use today”**

A major report on the global food system has found that less than one per cent of the world is eating a diet that’s good for the planet and human health. Switching to a healthier diet, however, could prevent up to 15 million premature deaths per year, while cutting global greenhouse gas emissions by up to 20 per cent.

These are the findings of a report by the 2025 EAT-*Lancet* Commission. The report brings together the expertise of nutritionists, climate scientists, economists, doctors, social scientists and agriculturalists from more than 35 countries around the world.

The Commission assessed the impact of the food system on human health and the environment, concluding that, in its current form, food production is jeopardising the planet and the long-term survival of the human race. It’s affecting the climate, land and water, disrupting the natural nitrogen and phosphorus cycles and causing pollution with pesticides and microplastics.

But, if the food industry changed to provide us all with healthier diets, we could address all of these environmental problems while also improving human lives.

“If everyone ate a healthy diet, we would be able to feed 10 billion people in 2050 with seven per cent less land than we use today,” Dr Fabrice DeClerck, chief science officer at EAT, told *BBC Science Focus*. “Never in the history of human food production have we occupied less of a resource to feed more people.”

The scientists estimated that 6.9 billion people were overconsuming food – specifically, meat, dairy, sugar and ultra-processed food – while 3.7 billion people struggled to access healthy food.

Their report recommends following the Planetary Health Diet (PHD), which prioritises fruits, vegetables, nuts, legumes and wholegrains.

On the PHD, half your plate would be filled with vegetables, fruits and nuts. Another 30 per cent would be wholegrains and the rest would be protein sources, prioritising legumes, such as beans, lentils and pulses.

Meat, fish and dairy foods are optional on the PHD (and there’s an upper limit), but the diet is flexible. For instance, you could eat up to 200g (7oz) of beef per week and still be within the guidelines.

DeClerck added that the diet can be tailored to the individual and adapted to people’s cultures and preferences. “If anything, we recognise that traditional diets are often better representations of healthiness,” he said.

POCKETS OF HEALTH

As for that one per cent already eating within the report’s recommendations, DeClerck said the scientists were not prepared to reveal exactly where these people lived, because there was a lot of variation within each country.

“But these people live in societies where you have liveable wages, where you have access to healthy diets, and they’re consuming those healthy diets,” he said. “The best examples of healthy diets often come from middle-income countries – particularly in the Mediterranean region, the Indian subcontinent and Southeast Asia.”

FOOD
Shifting global diets could prevent up to 15 million premature deaths per year

MONEY
Reshaping systems could deliver returns of $5 trillion a year through better health, restored ecosystems and climate resilience

ENVIRONMENT
Food systems currently account for roughly 30% of total greenhouse gas emissions globally

JUSTICE
The wealthiest 30% of people drive more than 70% of food-related environmental impacts

For people in those middle-income countries, the challenge was to avoid transitioning towards a more Western diet, said DeClerck.

DIVERSITY AND JUSTICE

Many of us find thinking about the climate crisis challenging, but DeClerck said that this report offered an “amazing” opportunity to improve human health and the climate at the same time.

“It encourages you to eat a more diverse range of foods, enjoy your own culture’s contribution, explore others and savour diversity. It’s a solution that’s not only good for your own individual health, it’s also a massive contribution to our collective planetary health.” This, he said, was “something to celebrate”.

Prof Johan Rockström, EAT-*Lancet* Commission Co-Chair and a co-author of the report, said: “The evidence is undeniable: transforming food systems is not only possible, it’s essential to securing a safe, just and sustainable future for all.”

Justice was also a key topic in the report, as it found that the wealthiest 30 per cent of people were driving more than 70 per cent of food-related environmental impacts.

“Those of us who are unhealthily overconsuming are impinging on the rights of others to have a safe environment,” said DeClerck.

The report called for urgent action to transform the global food system, for the sake of human health, justice and the environment.

HEALTH

Scrolling on the toilet could harm your health

The dirty habit may be more than just unhygienic

Using your phone while sitting on the toilet could have serious consequences for your health. According to a recent study published in *PLOS One*, the habit could increase your risk of developing haemorrhoids.

Otherwise known as piles, haemorrhoids are swollen veins in or around the anus that can cause pain and bleeding.

Scientists in the US conducted a survey of 125 adults whose gut health was being investigated with a colonoscopy (a procedure where doctors examine a patient's bowels using a camera inserted via their bottom).

The participants answered questions about their lifestyles and toilet habits, and the doctors who performed the colonoscopies assessed them for haemorrhoids.

In total, 66 per cent of the participants said they used their smartphones while on the toilet, mostly admitting to scrolling on social media or reading the news. Toilet scrollers were more likely to be younger than non-scrollers.

The scientists used statistical analysis to adjust for other factors linked to haemorrhoid risk, such as physical activity, age and diet.

They then found that people who brought their phones into the toilet had a 46-per-cent higher risk of haemorrhoids than those who didn't.

Time spent sitting on the toilet is likely to increase when you're distracted by your phone. In fact, 37 per cent of toilet scrollers in this study admitted to spending more than five minutes on the toilet per visit, compared to just 7.1 per cent of non-scrollers.

The scientists suggested that spending this extra time could increase pressure on scrollers' bottoms – including the anal tissues at risk of swelling up into haemorrhoids.

The study's senior author Dr Trisha Pasricha, an instructor of medicine at Harvard University, told *BBC Science Focus* that you shouldn't bring your smartphone to the toilet at all. "It distracts you from the task at hand."

If you do, she recommends using it to set a five-minute timer. But you'd be better off opting for old-school entertainment, such as a magazine, "that isn't expressly designed to make you lose all sense of time, like social media is."

AGEING

Spatial awareness could reveal dementia risk

Possible link suggested by results of small study

A new study suggests that subtle navigation difficulties could be an early sign of Alzheimer's disease, showing up before standard memory tests detect any decline.

An international team of researchers tested approximately 100 older adults in a virtual reality (VR) experiment designed to challenge the participants' sense of direction.

Their findings show that people with subjective cognitive decline (SCD) – a condition in which people feel their memory is worsening despite normal results on clinical tests – were less accurate at orienting themselves than their healthy peers.

"People with SCD are known to be at an increased risk of developing Alzheimer's later in life," said Prof Thomas Wolbers, one of the study's authors.

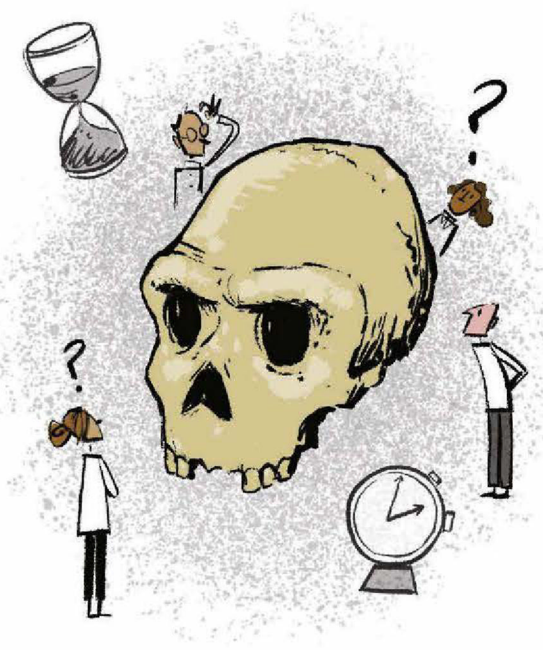
As part of the experiment, participants, aged 55–89, were asked to don VR headsets and

ILLUSTRATIONS: ADAM GALE

ARCHAEOLOGY

60-year mystery of the fossil skull that baffled scientists may finally be solved

The Petralona skull was discovered in Greece in 1960, yet its origin has perplexed experts – until now



A strange, fossilised skull found in a Greek cave more than 60 years ago may finally be giving up some of its secrets. New analysis, detailed in a recently published study, suggests it's at least 300,000 years old, dating it back to a time when *Homo sapiens* were still in Africa and the first Neanderthals had appeared in Europe.

Known as the Petralona skull, it has long caused headaches for palaeoanthropologists, who struggled to determine where it fits into the human family tree.

The fossil lacked distinct features of well-known human species and was found in isolation, embedded in a cave wall with no surrounding artefacts or animal bones that could be dated.

"As soon as I saw it and measured it, I knew that it wasn't Neanderthal or *Homo erectus*," Prof Chris Stringer, a palaeoanthropologist at the Natural History Museum in London and one of the study's co-authors, told *BBC Science Focus*. "It was something different."

Stringer long suspected that the specimen belonged to another of our cousins: *Homo heidelbergensis*, a robust human species that lived hundreds of thousands of years ago across Africa, Europe and possibly Asia.

Without a firm date for the skull, however, it was difficult to know where it fit into the human story.

The new analysis applied uranium-series dating to the calcite coating that had developed on the fossil.

Calcite is one of the most common mineral forms of calcium carbonate, and often precipitates on (or within) fossils when mineral-rich groundwater infiltrates their pores and cavities. But with the help of uranium-series dating (a method that detects how much uranium breakdown has occurred in the calcite), it can be used to help calculate a fossil's age.

The results of this new analysis of the fossil skull point to it having an age of around 286,000 years. Yet there is still some uncertainty. If the calcite was deposited quickly on the fossil, then the estimate is a good bet, Stringer said. "But if the fossil had actually lain around in the cave before the calcite got on it, then that age is only a minimum age."

Nevertheless, the Petralona skull's timeline and shape fit with another famous *H. heidelbergensis* skull from Kabwe, Zambia, dated to around 300,000 years ago. This strengthens the view that *H. heidelbergensis* were widespread and long-lived, overlapping with other human species rather than directly giving rise to them. They were almost certainly contemporaries with modern humans – at least for a time.

"The results of this new analysis of the fossil skull point to it having an age of around 286,000 years"

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walk across an empty, featureless plain in the virtual environment. Their task was to follow a floating ball along a winding path and then point back to where they had started. They then had to turn and face the original direction they had been facing at the start.

Although everyone performed within the normal range on conventional memory and thinking tests, those with SCD consistently made larger errors in the navigation task.

"These orientation difficulties didn't arise from movement dynamics, such as walking faster or looking at the ground more often while walking," said Dr Vladislava Segen, one of the study's authors. "The causes of the imprecise orientation were not related to motion, but cognitive in nature."

According to the researchers, the SCD group may have performed poorly due to a "memory leak", whereby participants struggled to keep track of the sequence of past positions needed to update their location in real time. The researchers believe this may stem from dysfunction in grid cells – specialised neurons that build a mental coordinate system of our surroundings.

The team hopes this kind of spatial testing could one day complement existing tools for diagnosing Alzheimer's.

SPACE

Rogue planet is now the fastest-growing world ever seen

The hungry planet is swelling up like a star

A sudden growth spurt has led to a 'rogue' planet becoming the fastest-growing planet astronomers have ever seen.

The planet unexpectedly began gorging on the gas surrounding it and is now swallowing down six billion tonnes (2.2 trillion pounds) every second, according to a new study. That makes it the hungriest world on record.

The planet, named Cha 1107-7626, is around 5–10 times the mass of Jupiter and is located 620 light-years away. Being a rogue planet, it doesn't orbit a star, but instead floats freely through the cosmos on its own.

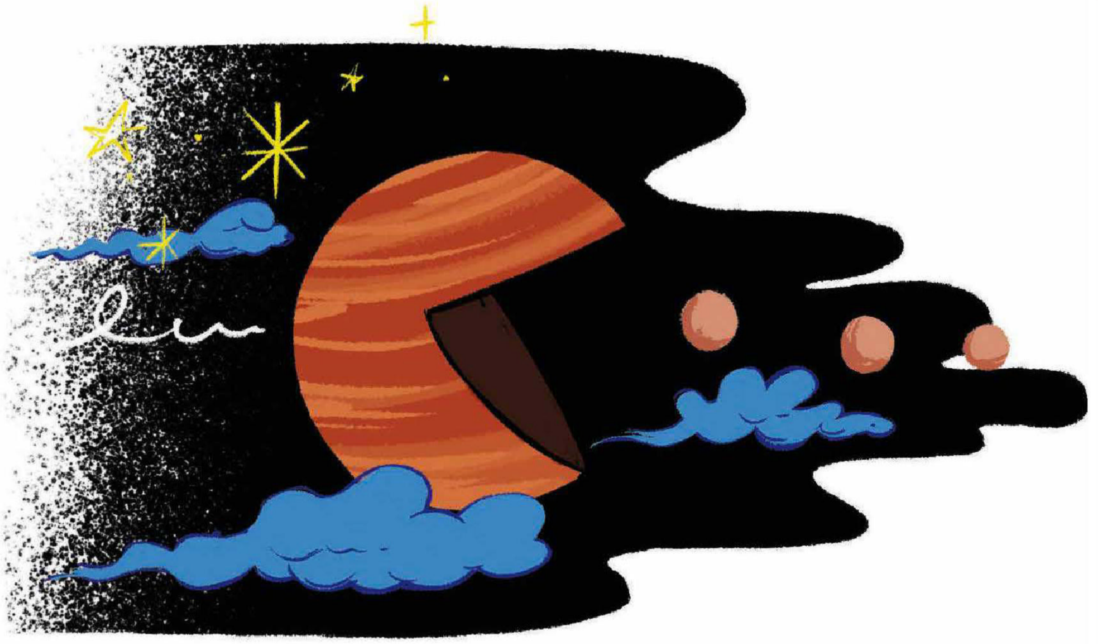
"People may think of planets as quiet and stable worlds, but with this discovery we see that planetary-mass objects freely floating in space can be exciting places," said Dr Victor Almendros-Abad, an astronomer from the National Institute for Astrophysics, in Italy, who led the study.

Cha 1107-7626 is surrounded by a disc of gas and dust that's constantly falling onto the planet's surface, allowing it to grow through a process known as accretion.

Over the last year, astronomers noticed the planet was growing at an increasingly faster rate, feasting on more and more gas. By August 2025, it was gathering eight times more gas than it had done just a few months before, making it the strongest-ever accretion episode for a planetary-mass object.

Where rogue planets come from is still a mystery, but the discovery seems to suggest this one is forming more like a star than a planet.

"The idea that a planetary object can behave like a star is awe-inspiring and invites us to wonder what worlds beyond our own could be like during their nascent stages," said Dr Amelia Bayo, an astronomer for the European Southern Observatory, who also took part in the study.



DIET

Taking common vitamin D supplements may actually lower your vitamin D3 levels

Scientists are 'astonished' that the supplements could actually make deficiency worse

Taking vitamin D2 supplements could actually reduce the levels of vitamin D3 in your body, according to a surprising new study published in *Nutrition Reviews.*

The finding comes just as much of the Northern Hemisphere heads into autumn – the time when health experts recommend people start supplementing with vitamin D.

Vitamin D is essential for regulating calcium and phosphate in the body, which keeps bones, teeth and muscles healthy. Deficiency is common worldwide: in the US, rates are as high as 31 per cent among non-Hispanic Black adults.

But not all vitamin D is created equal. It comes in two main forms: Vitamin D2, which is found in plants and mushrooms; and Vitamin D3, which is produced in the skin when

ILLUSTRATIONS: ADAM GALE

exposed to sunlight, but is also found in animal products such as oily fish.

During the summer, people living in higher latitudes can usually make enough vitamin D through sunlight exposure. In autumn and winter, however, the Sun doesn't rise high enough for this to happen.

"People often think that on a sunny day in October, November and December they'll make vitamin D," Prof Susan Lanham-New, head of the department of nutritional sciences at the University of Surrey and a co-author of the study, told *BBC Science Focus*. "But they won't make any."

To explore the effects of vitamin D2 supplementation, Emily Brown, a PhD research fellow in Lanham-New's group, analysed 20 randomised controlled trials. In 18 of them, participants who received vitamin D2 had lower levels of vitamin D3 compared with placebo or control groups.

"We don't want people to think vitamin D2 is in any way dangerous. It's not," Brown said. "Vitamin D2 still raises your total vitamin D levels, but we think vitamin D3 is preferential."

Previous research has shown that vitamin D3 is converted more efficiently into the active form of the vitamin than vitamin D2, suggesting it may be a better choice for supplementation, particularly during the darker months.

"We were really astonished that when you give D2, the D3 levels drop below even the placebo," Lanham-New said. "There's a lot more work that we now need to do because the long-term impact could be that you don't reach the required vitamin D levels or, dare I say it, you could make the situation worse."

"People often think that on a sunny day in October, November and December they'll make vitamin D. But they won't make any"

EVOLUTION

We finally know why men don't live as long as women, say scientists

Males that spend less energy on sex and more on raising their children tend to live longer

A group of researchers at Germany's Max Planck Institute for Evolutionary Anthropology has been trying to find out why men tend to live shorter lives than women.

Their study, published in *Science Advances*, examined over 1,000 animals to track down common evolutionary traits in species where one sex tends to outlast another.

They discovered that although genetics does seem to play a part, monogamy and child-rearing also affect lifespans.

In humans, women live an average of 5.4 years longer than men, but we're not the only species where that happens. In fact, in 72 per cent of mammals, females outlast their male counterparts by an average of 12 per cent.

In birds, however, it's males that tend to live longer – by an average of five per cent in the majority of species.

One of the leading explanations is chromosomes. In mammals, females have two X chromosomes, while males have one X and one Y chromosome.

With only one X chromosome, any harmful mutations are more likely to have an impact, as there isn't a second copy to compensate. Meanwhile, the 'toxic Y' hypothesis suggests that the Y chromosome may carry potentially harmful effects on health and ageing.

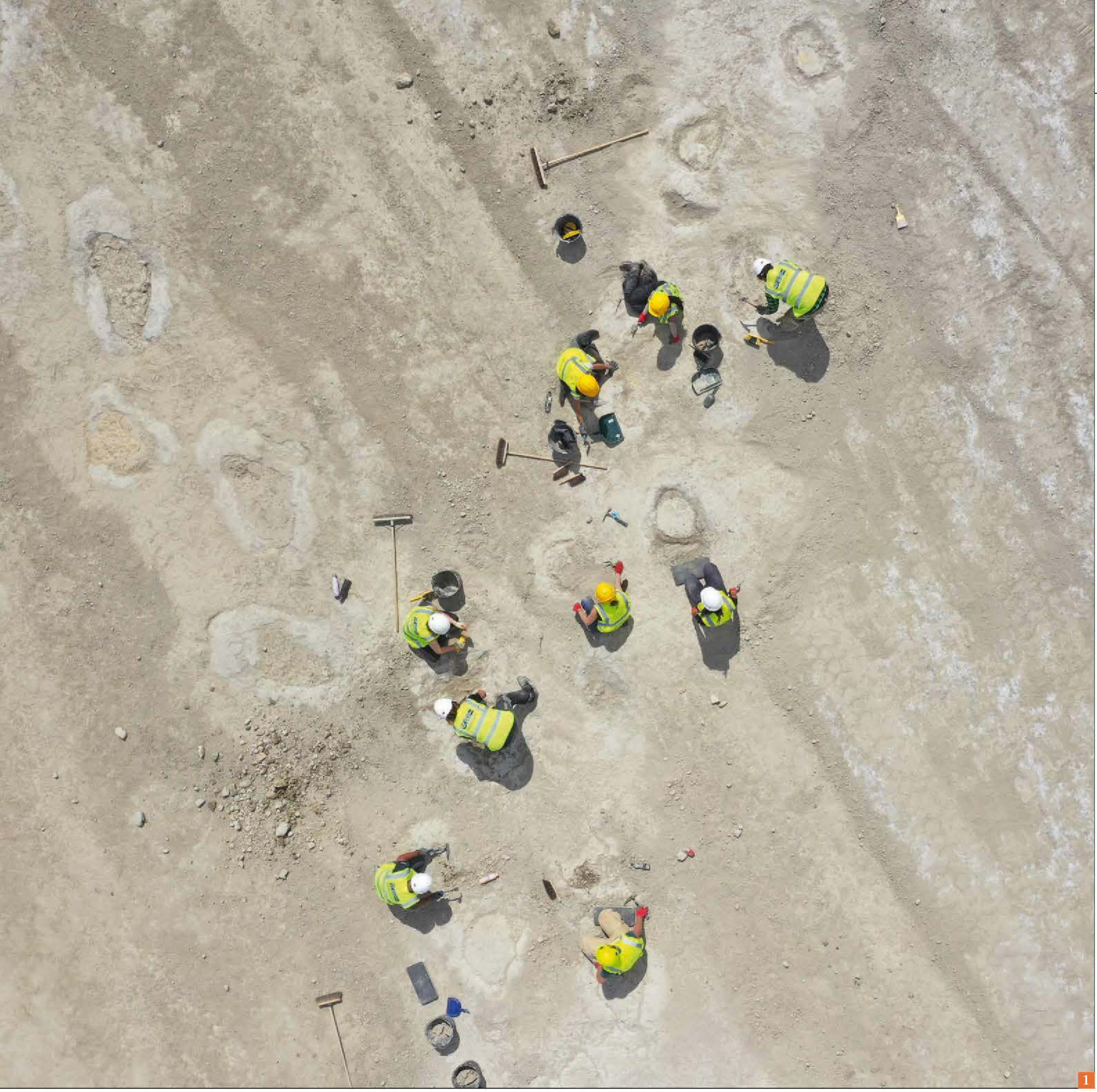
As for birds, males have two Z chromosomes, while females have one Z and a truncated W. Yet, since not all bird species see longer lifespans in males, there must be another factor at play. The researchers think that might be mating, as birds generally tend to be monogamous, while only a small percentage of mammals stick with one partner.

"Males often pay the price for reproduction," Dr Johanna Stärk, the study's lead, told *BBC Science Focus*. "To secure mates, they invest in traits like manes or antlers and spend energy fighting rivals, which increases their risk of injury and shortens their lifespan."

The researchers also found that child-rearing seems to have an impact. Ask any parent and you'll know that bearing and raising children takes a lot of energy, so you might expect the sex responsible for child-rearing to pay the price with a shorter lifespan.

"Instead, we found the opposite – the caregiving sex often lives longer," said Stärk. "This seems especially true in species like primates, where the young remain dependent for a long time."

Based on this, it would seem that being a loyal partner and a good parent hold the keys to a longer life for males – at least from evolution's point of view.



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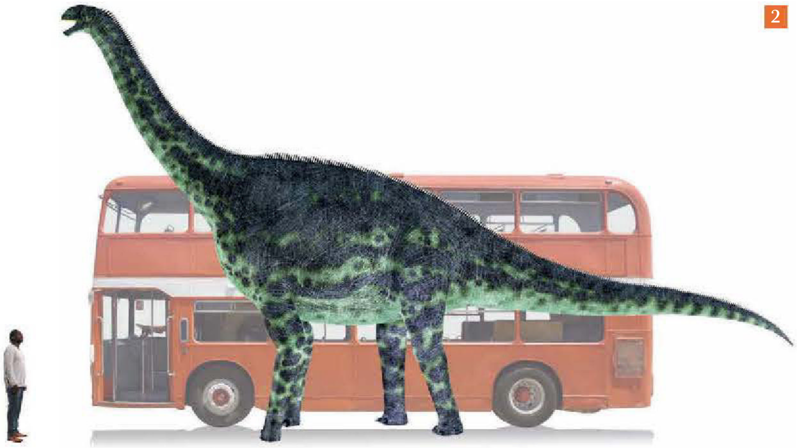
1. Millions of years ago, the UK was much closer to the equator and it's likely this site was something like a shallow lagoon. Today, remnants of ancient marine life, including seashells, squid-like creatures and sea urchins can be found all over the floor at Dewers Farm Quarry.

2. Sauropods could grow to about 18m (59ft) in length, almost twice as long as a double-decker bus. The researchers think that the Oxfordshire dinosaurs were likely of the genus *Cetiosaurus*, which they know lived in the area. Their name translates as 'whale lizard'.

3. At about 1m (3.3ft) across, the footprints are about twice the diameter of a modern-day elephant's. With so many prints to study, the scientists could analyse how the dinosaurs would have moved. Using 3D models, they worked out one sauropod was strolling along at two metres per second when it left its prints – about the same speed as a human walking quickly.

4. The scientists also discovered a *Megalosaurus*'s footprint at the quarry. The two-legged carnivore left three-toed footprints and was much smaller than the sauropods, but still a massive 6m (20ft) in length.

2



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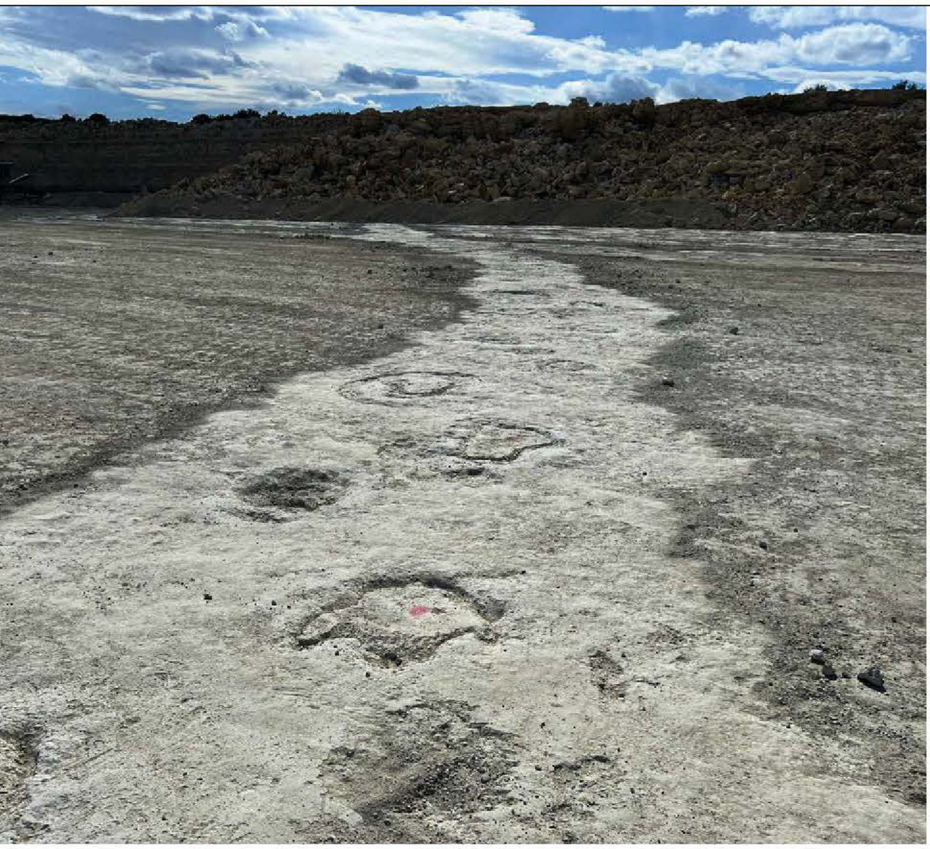

PALAEONTOLOGY

Dinosaur 'superhighway' unearthed in the UK

The find could change our understanding and portrayals of dinosaurs

One of the longest dinosaur paths has been discovered in an unlikely location.

'Jurassic trackways' are rare in the UK, yet in a quarry in Oxfordshire, scientists have revealed a dinosaur 'superhighway' full of footprints left by sauropods – the largest animals to ever walk on land.

Dewars Farm Quarry has been known for its dinosaur footprints since the 1990s. But when scientists discovered 200 footprints in 2024, they suspected there might be even more to be found. Using controlled explosions, they discovered the ancient footprints buried under tonnes of limestone rock.

The path the explosions revealed is 220m (722ft) long – one of the longest continuous trackways ever found.

"We're uncovering something that people have never seen before," palaeontologist Prof Kirsty Edgar of the University of Birmingham told *BBC News*. "It's so rare to find something this big."

Tracks like these require very specific conditions to be preserved. Researchers think the prints were made as the sauropods' heavy feet sank into mud, which was then likely baked by the Sun and later covered with sediment from a storm that blew in soon after.

The scientists say that skeletons can only tell you so much about dinosaur movement and behaviour. A discovery like this could provide fresh insight into their gaits and lead to more accurate portrayals on TV and in movies.

UNIVERSITY OF BIRMINGHAM, DR EMMA NICHOLLS/OXFORD UNIVERSITY MUSEUM OF NATURAL HISTORY X2, GETTY IMAGES X2

DOGS

Your dog really could be addicted to their ball, says new study

'Ball junkies' are real and some will go to great lengths to get their toy fix

Dogs love their toys, but can they love them too much? According to a new study, yes – our canine companions' playful spirits can sometimes take a turn towards addiction.

Recently published in *Nature Scientific Reports*, the study found that some dogs act in ways that parallel humans with behavioural addictions to activities like gambling or playing video games.

"The term 'ball junkie' is commonly used in the dog training world, implying addiction. But it has never been assessed whether addiction criteria may indeed apply to dogs," Prof Stefanie Riemer from Austria's University of Medicine Vienna, one of the study's authors, told *BBC Science Focus*. "After all, just being highly motivated, or very toy-focused does not imply an addiction."

The team behind the study brought 105 toy-obsessed dogs in for a series of tests to understand how they interacted with their toys, especially when two other powerful motivators – food and their owner's attention – were also on offer.

In the end, 33 dogs showed signs of addiction-like behaviour. They would focus on the toy, trying to reach it when it was stored inside a closed box or placed on a high shelf. This happened even when there was food on offer or their owners tried to engage with them by playing a game of tug-of-war.

"Two dogs actually managed to destroy the box containing their toys, illustrating the intensity of their motivation," Dr Alja Mazzini, the study's lead, told *BBC Science Focus*. "It wasn't something we encouraged or expected, but it was simply a striking example of how far some dogs were willing to go to access the object of their desire."

When the toys were removed from the room, some of the dogs were unable to settle. They walked around the room, searched for their toys and still had elevated heart rates after 15 minutes.

"While the majority of dogs enjoy playing with toys, only a few develop an excessive fixation on them," said Mazzini.

"This is a pattern that mirrors human behaviour, where many people are able to engage in potentially addictive activities such as gaming or gambling without consequences, while only a few develop a genuine addiction."

> "Two dogs managed to destroy the box containing their toys"

ABOVE "Addicted? No, I can stop playing with this ball anytime I like"

GETTY IMAGES

2000T

JACK RYDE 100 YEAR ANNIVERSARY LIMITED EDITION

Limited to 2,000 numbered pieces, the Jack Ryde 100 Year Anniversary Limited Edition 2000T features blue saddle leather corner guards and matching leather lifting handles, embroidered signature, married with rich bronze accents and distinct signature plate celebrating Jack's legacy. Discover more at **hastens.com** or visit your nearest **Hästens store**.

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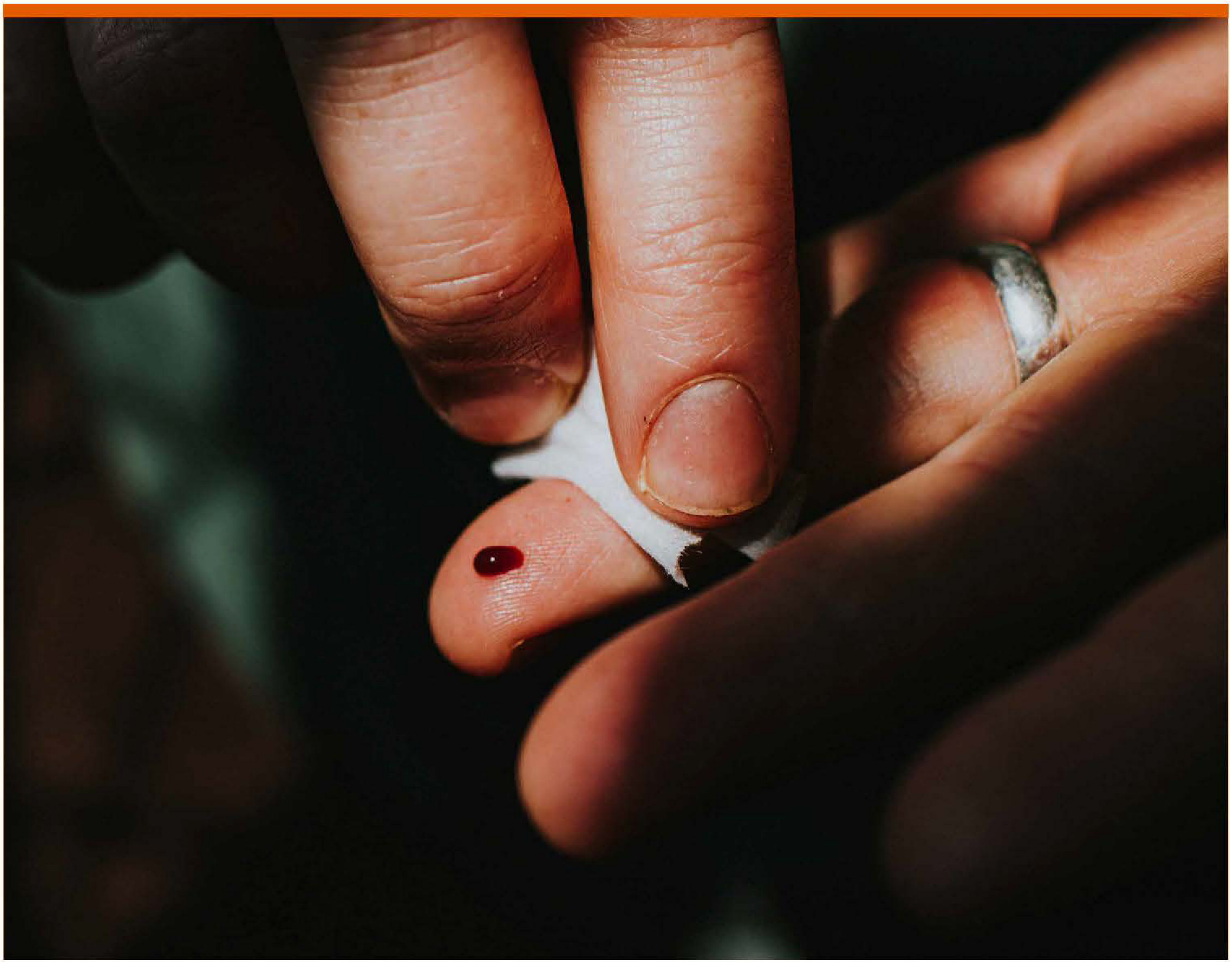
REALITY CHECK

THE SCIENCE BEHIND THE HEADLINES



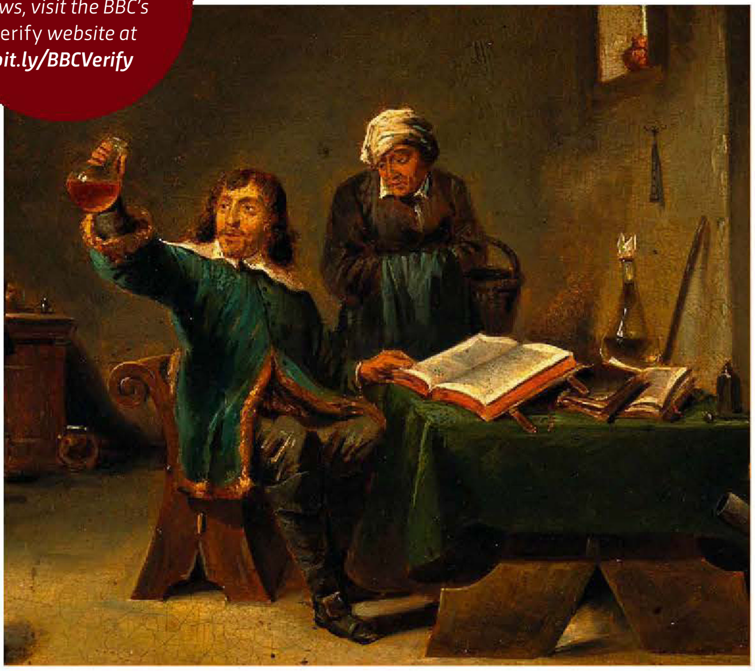
REVIEW

There's another diabetes in town, here's how to recognise it

Misdiagnosis rates for this rare type of diabetes could be complicating treatment for patients

BBC

For more fact-checking news, visit the BBC's Verify *website at* ***bit.ly/BBCVerify***



ABOVE In the past, medical practitioners would examine a patient's urine to diagnose diabetes, as depicted in this artwork from the 17th century

OPPOSITE A finger-prick test is one way for someone with diabetes to check their blood glucose levels

You might be surprised to learn that there are more than two types of diabetes. We're all familiar with types 1 and 2, but you may have seen another one – type 1.5 – making headlines recently.

Type 1.5 diabetes, or latent autoimmune diabetes in adults (LADA) to use its official name, is not a new form of diabetes, but it is somewhat rare. It's been medically recognised as a distinct form of diabetes since 1993 and accounts for 3–12 per cent of all cases of diabetes in adults.

But as LADA shares similarities with its two, more famous, cousins, it's also trickier to identify. Hence, it's often misdiagnosed. As many as 14 per cent of people diagnosed with type 2 diabetes may actually have LADA, which is why it's been in the headlines of late.

Like type 1 and 2 diabetes, LADA is classed as a disorder of too much sugar, or glucose, in the blood.

When this occurs, it doesn't matter what kind of diabetes you have, the symptoms remain the same: extreme thirst, frequent visits to the toilet, fatigue and unexplained weight loss. Diabetes UK, the leading UK charity for the disease, describe these as 'the four Ts': thirst, toilet, tired and thinner.

> **"The onset of LADA is therefore more similar to type 2 diabetes"**

Once these symptoms appear, it's important to seek a diagnosis as soon as possible. Untreated diabetes can lead to a host of severe complications, impacting the kidneys, eyes and feet, as well as the nerve cells in your body.

But if the symptoms for these diseases are all the same, how can experts tell which kind of diabetes you really have? And how does it affect the way you treat it?

HOW TYPE 1.5 DIABETES COMPARES TO TYPES 1 AND 2

Diabetes, in its many forms, has afflicted humans throughout history. A peculiar symptom often alerted healers in Ancient Greece, India and Egypt to the condition: the patient's urine smelled sweet. Hence the scientific name Diabetes mellitus, taken from the Greek word 'diabetes', which means to pass through, and the Latin word 'mellitus', meaning sweet.

This aromatic side effect is caused by a build-up of glucose in the body. Glucose is meant to be broken down by a hormone called insulin, which is produced in the pancreas and converts glucose into a form that can be used as an energy source in cells.

A build-up of glucose happens when a person's body either isn't producing enough insulin (as is usually the case in type 1 diabetes) or isn't reacting to insulin the way it should (as in type 2 diabetes).

In either case, a lot of extra strain is put on the kidneys, which try to reabsorb the excess glucose floating around in the blood. When the kidneys can't remove any more glucose, the excess sugar spills into the urine, offering a sign of the disease.

LADA shares a lot of similarities with these two main types of diabetes, but it has more in common with type 1 than type 2.

One of the main reasons people with type 1 diabetes don't produce enough insulin is that their immune system attacks the cells in their pancreas that make the insulin. When a substantial number of these cells are lost, they can no longer make enough insulin to keep glucose levels in check.

The same is true in LADA – a swarm of immune cells attacks the pancreas, leading to a drop in insulin production, which causes glucose levels to steadily increase. The two differ, however, when it comes to how long the disease takes to have an effect.

Type 1 diabetes takes effect rapidly, and is likely to be diagnosed in childhood or in a clinical emergency. LADA has a slower burn (hence the 'latent' in latent autoimmune diabetes) and doesn't kick in until adulthood, commonly occurring in people above the age of 30 (hence the 'in adults').

As you age, a protective scaffold known as the peri-islet basement membrane forms around the insulin-making cells in your pancreas. Current thinking suggests that this scaffold helps to keep your immune cells from attacking your pancreas.

By the time that "autoimmunity starts in LADA, the pancreatic [cells] have already formed into larger structures and they're better protected. They're better able to resist the immune attack," says Dr Sarah Richardson, professor of cellular biomedicine at the University of Exeter.

The onset of LADA is therefore more similar to type 2, which usually rears its head during adulthood. It's because of this similarity that up to 14 per cent of people diagnosed with type 2 diabetes are actually misdiagnosed cases of LADA.

GETTY IMAGES, ALAMY

> “The first and most important thing doctors can do to differentiate the disease is to check for antibodies”

WHY MISDIAGNOSIS MATTERS

The first line of treatment for type 2 diabetes is a medication called metformin. Given in tablet form, metformin works in two ways. Not only does it stop the liver from producing too much glucose, but it also increases the body’s sensitivity to insulin.

There isn’t, however, a definite, agreed-upon way of managing LADA yet. Metformin can be prescribed, as can insulin replacement therapy. If a person is misdiagnosed with type 2 diabetes when they really have LADA, they could be put on a course of metformin when they really need insulin.

The longer a person takes the wrong medication, the higher their glucose levels can climb. Over the longer term, high glucose levels increase the risk of heart disease and stroke, eye problems (retinopathy), foot problems (which can range from infections and ulcers to a loss of sensation), kidney disease (nephropathy) and nerve disease (neuropathy). A 2018 study published in *Diabetic Medicine* found that people with LADA were more prone to developing severe neuropathy than people with type 2 diabetes.

LADA can also cause damage to small blood vessels, known as microvascular disease. A 2020 study found that implementing strict glucose control from the time a person was diagnosed with LADA could significantly reduce the later risk of microvascular disease.

Cutting down rates of misdiagnosis is therefore crucial for people with LADA, and thankfully, there are a few key ways to tell it apart from type 1 and type 2 diabetes.

by **PROF CRAIG BEALL**

Craig is the Diabetes, Obesity and Metabolic Health Research Theme Lead at the University of Exeter.

The first and most important thing doctors can do to differentiate the disease is to check for antibodies. These falsely tell immune cells to attack the insulin-producing cells in the pancreas. The presence of at least one antibody indicates autoimmune diabetes. If antibody levels are high, LADA may progress more quickly.

The same is true if multiple antibodies are found. In this case, insulin injections should be the first course of treatment, giving immediate and better control over glucose levels.

Doctors can also directly check a person’s insulin production. This is done by measuring a protein in their blood, called c-peptide, that’s made during pancreatic insulin production. Higher levels of c-peptide can be treated with metformin tablets, and very low or undetectable levels mean that insulin should be started immediately. In LADA, c-peptide levels decline with time – the current recommendation is to get them checked every six months.

A person’s body type can also be an indicator of LADA. If someone develops an increase in the ‘four Ts’ – thirst, toilet, tired and thinner – but has relatively low fat levels or body weight, they’re generally more likely to have LADA than type 2 diabetes. People with LADA also typically have better cholesterol, which can be used to narrow down a diagnosis.

Once the correct diagnosis has been made, LADA can be treated appropriately. It’s getting to this place, with the help of a medical professional, that’s critical.

RIGHT Diabetes medication comes in various forms. Insulin, for example, (typically a treatment for type 1 diabetes) can be administered by injection or, as seen here, via an automatic pump

GETTY IMAGES, RED BULL CONTENT POOL

SINGAPORE
SINGAPORE AIRLINES

ABOVE When the F1 championship returns to Singapore in October 2026, the cars will be running on a new type of sustainable fuel

ANALYSIS

Cleaner, greener fuels are coming to Formula 1. Here's what that means

The 2026 F1 championship will be raced using 'sustainable' fuels. But how much of a difference will it actually make to the sport's carbon footprint?

Next year, Formula 1 (F1) will pull into the pits for one of its most ambitious overhauls yet – trading fossil fuel for a 100-per-cent sustainable alternative. It's part of a broader effort to meet new environmental rules and to prove the sport can, in F1's words, "keep us driving without building new cars."

But not everyone's convinced. With fuel from races making up less than one per cent of the sport's total carbon footprint, experts say F1 has far bigger environmental problems to solve. So, what are they and how can they be tackled?

FRESH FUEL

In 2020, F1's governing body, the Fédération Internationale de l'Automobile (FIA), set a deadline for race cars' engines to run on 100-per-cent sustainable fuels from 2026 and for the sport to be carbon neutral by 2030.

The transition began in 2023 and 2024, when Formula 2 and Formula 3 (F1's feeder racing series) started using 55-per-cent 'sustainable bio-sourced fuel', before switching to 100-per-cent 'advanced sustainable fuel' in 2025.

Now, F1 has developed its own 'sustainable' fuel for 2026. This fuel has been designed specifically for the hybrid engines in today's F1 cars, which rely on both an internal combustion engine and two electric motor-generators.

F1 says the new fuel won't add to the overall amount of carbon in the atmosphere. Instead, the carbon used in the new fuel will be extracted from existing sources – including household waste and non-food biomass – or captured as carbon dioxide directly from the atmosphere.

This will then be used to make a synthetic fuel – an artificially-produced fuel designed to do the same job as the fossil fuel-derived petrol that's currently being used. In the long term, the FIA says that F1, 2 and 3 will all eventually adopt this "fully synthetic hybrid fuel".

But it won't just be for race cars. F1 describes the new fuel as a 'drop in', meaning it'll work not only with existing combustion engines, but also with fuel distribution networks. In other words, the fuel that will be powering F1 cars in 2026 is one that you could soon be pumping into your car at your local petrol station.

WHAT'S MEANT BY SUSTAINABLE?

The widespread use of the word "sustainable" has led experts to scrutinise the claims being made by F1, however. Dr Paula Pérez-López specialises in environmental and social sustainability of products at the MINES ParisTech Centre for Observation, Impacts and Energy. She explains that for a product to fulfil the definition of sustainable, it must meet specific environmental, social and economic criteria and consider these aspects in every part of its supply chain.

"The term 'sustainable' is definitely not the same as 'low-carbon'. A product or process may be low-carbon while having high emissions of other pollutants, thus being 'unsustainable'."

The FIA appointed the Zemo Partnership, a UK-based non-profit partnership, to develop the Sustainable Racing Fuel Assurance Scheme. This is a third-party scheme to certify the use of sustainable racing fuel, ensuring it meets the FIA's regulatory standards. This requires the fuel to "be made from a minimum of 99-per-cent advanced sustainable components certified to have been derived from a renewable source of non-biological origin, municipal waste or non-food biomass." In layman's terms, the new fuel must be either synthetic, made from waste products, or derived from materials not intended for human or animal consumption, such as specially developed algae.

The new fuel must also meet the qualities outlined by the European Union's Renewable Energy Directive III and Delegated Act.

Fraser Browning, founder of Kurb Carbon, a company that advises businesses on how to minimise their climate impact, says that, if managed responsibly, the new fuel will deliver a genuine decarbonisation initiative.

"The bigger question is around F1's overall impact," he says. "Why has F1 decided to take this route into synthetic fuels? Because it's a fundamental part of its sustainability journey? Or is it just a box-ticking exercise?"

Browning points out that innovation in motorsports has previously led to great leaps forward in sustainable motoring as a whole. In 2020, for example, Mercedes announced that its hybrid technology for F1 cars would be adapted into its road cars. Earlier this year, the German car maker also unveiled a new battery that could extend the range of its electric road cars by 25 per cent.

"I believe that we wouldn't have hybrid vehicles in the form we do today, and as quickly, if it hadn't been for input

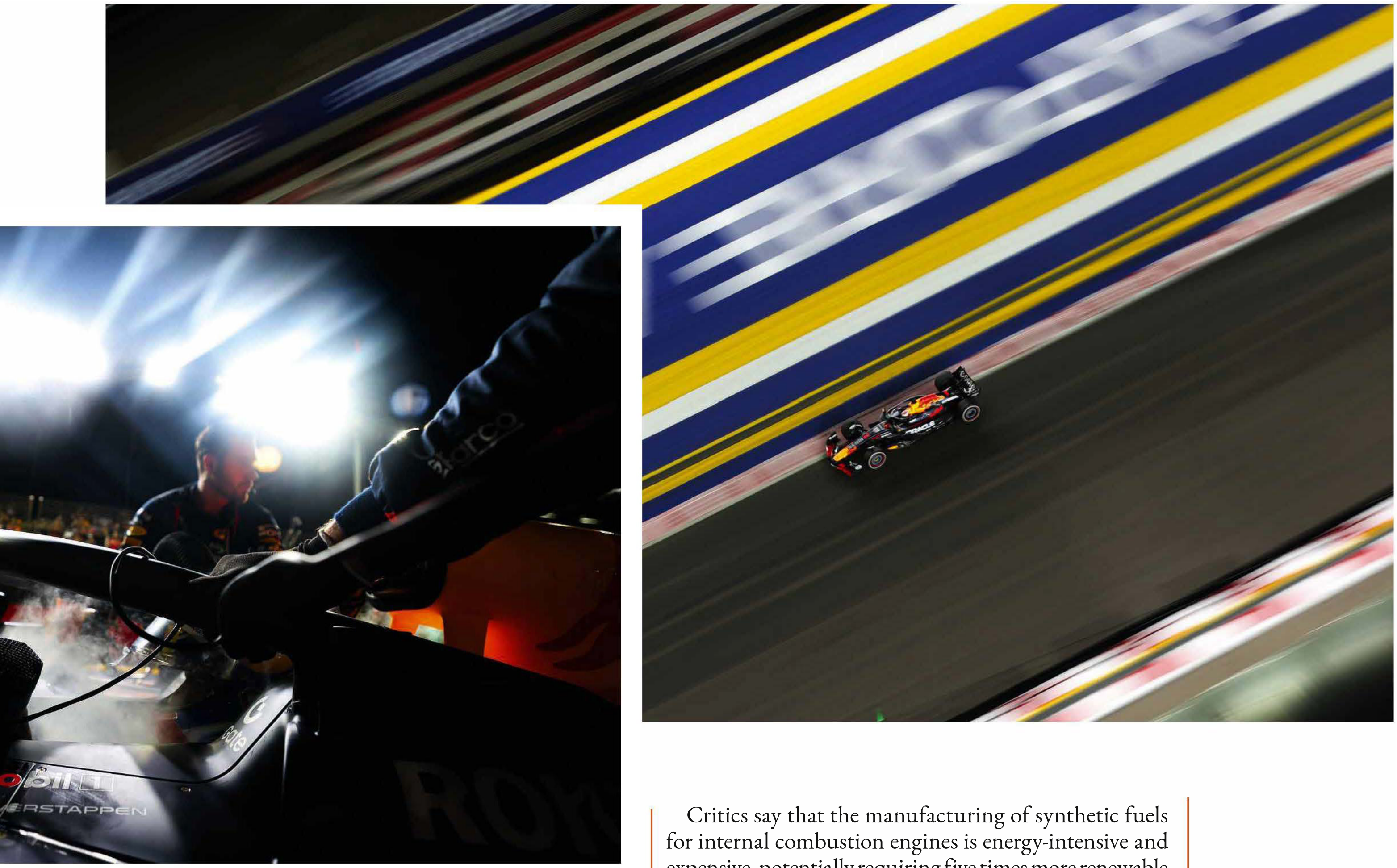
"By the end of 2024, F1's total carbon emissions were reduced by 26 per cent compared to the 2018 baseline"

ABOVE LEFT Cars competing in the Formula E championship are battery-powered

ABOVE A raft of new F1 regulations, besides the change of fuel, will come into effect in 2026

ABOVE RIGHT The race cars' fuel accounts for less than one per cent of F1's total carbon footprint

GETTY IMAGES, RED BULL CONTENT POOL



from sporting innovation," he says. "But it has to be done in a transparent and responsible way."

Beyond the switch to synthetic fuels, F1 has also made efforts to reduce its carbon emissions in other areas. Travel and logistics make up two-thirds of F1's carbon footprint, with teams, heavy mechanical kit and fans travelling vast distances between races each year.

According to F1's latest Impact Report, in 2024, changes were made to the F1 calendar to reduce the freight distance between events. For example, the Japanese Grand Prix was rearranged to take place in April, grouping it with other races in the Asia-Pacific region.

F1 also expanded the use of biofuel in the trucks used to transport its freight across Europe. Changes like these led to a nine-per-cent reduction in carbon emissions in the logistics side of F1.

By the end of 2024, total carbon emissions were reduced by 26 per cent compared to the 2018 baseline, but F1 admits that it has "major milestones to achieve, including further investment in alternative fuels across our operations and updating our logistics network to maximise efficiency."

SYNTHETIC FUELS VS ELECTRIC VEHICLES

What about the claims that F1's new synthetic fuel will be a drop-in that could be used for everyday cars driving on public roads? Could it offer a more sustainable alternative than electric vehicles (EVs)?

Critics say that the manufacturing of synthetic fuels for internal combustion engines is energy-intensive and expensive, potentially requiring five times more renewable electricity than running a battery-powered electric vehicle.

Currently, 96 per cent of hydrogen used to make these fuels in the EU is produced from natural gas, a process that emits considerable amounts of CO_2. Currently, renewable hydrogen is also more expensive than fossil-based hydrogen.

"Obtaining pure, concentrated CO_2 is a major challenge," says Gonçalo Amarante Guimarães Pereira, professor at the State University of Campinas, in São Paulo, Brazil and co-author of a study that compared biofuels with pure electric vehicles.

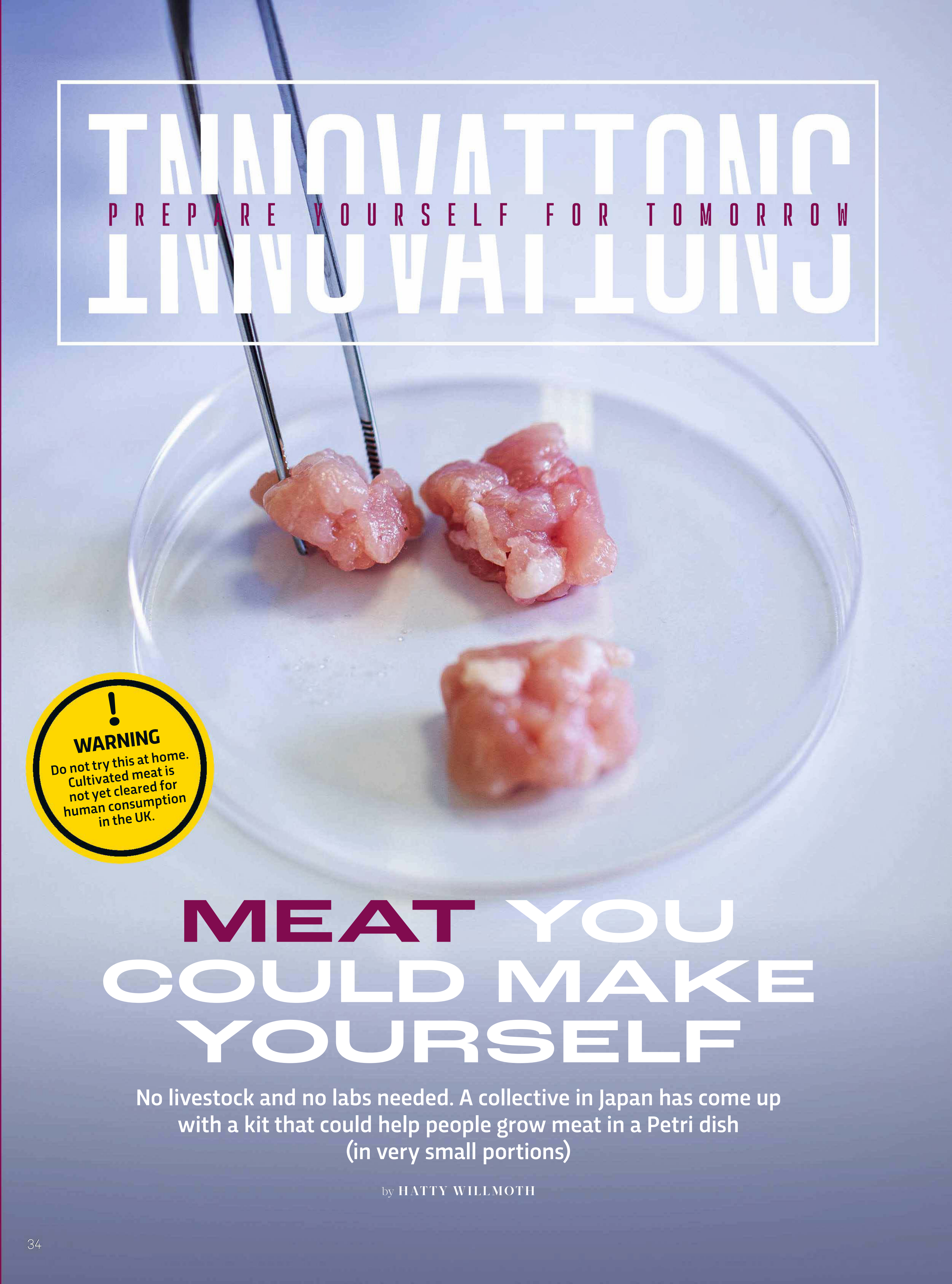
"There's a technology called Direct Air Capture that can achieve this, but to reach a concentration of 100 per cent requires enormous energy consumption. Estimated costs range from $500–$1,200 (approx £375–£895) per tonne, making e-fuel at least four to eight times more expensive than driving an electric car."

Browning agrees that EVs provide a better low-carbon option in the long run. "Their maintenance and in-use emissions are far lower," he says.

"Synthetic fuels, when processed responsibly, may provide a lower impact overall, but I've yet to see a comprehensive life cycle assessment across multiple sustainability metrics that provides a detailed answer to this dilemma."

In other words, until every part of the system that produces a synthetic fuel can be reliably proven to have a positive impact on the environment, the jury's still out on how much good they'll actually achieve. **SF**

by **ANA SANTI**
Ana is a journalist who specialises in sustainability and climate change, and the author of Three Things to Help Heal the Planet *(Welbeck Balance, 2022).*



WARNING
Do not try this at home. Cultivated meat is not yet cleared for human consumption in the UK.

MEAT YOU COULD MAKE YOURSELF

No livestock and no labs needed. A collective in Japan has come up with a kit that could help people grow meat in a Petri dish (in very small portions)

by HATTY WILLMOTH

Growing your own fruit and veg is nothing new. But what if you could grow your own meat in the comfort of your own home? That's what a company in Japan wants to make possible.

The Shojinmeat Project has developed a method for people to do just that.

It's called cultivated meat, and it involves taking a few animal cells and helping them grow in a tank, or bioreactor. It's real meat, but it doesn't come via the typical farm and slaughterhouse route.

Yuki Hanu, the founder and director of the Shojinmeat Project, describes the company as a not-for-profit citizen science project, which aims to enable "restaurant chefs or hobbyists to grow designer meat in their premises".

"We've been successful in establishing an entirely DIY version of animal cell culture protocols," he says, explaining that the project provides people with instructions and a shopping list of items for cultivating small amounts of chicken at home.

The project is, at present, for educational and research purposes only, however, not for food production or consumption, which remain tightly regulated. It's also important to note that cultivated meat produced outside approved labs can't legally be sold or eaten in the UK.

A spin-off of the Shojinmeat Project, called IntegriCulture, has also developed a bioreactor system that people can buy and use in a home or restaurant. It's pre-built, so less DIY than the Shojinmeat kit, but Hanu says it's still far cheaper than using a lab-grade bioreactor, like those used to make commercial lab-grown meat.

HOW IT WORKS

So, you've decided you want to grow your own chicken. First, you'll need to go shopping.

The Shojinmeat Project has a list of items you should be able to buy in supermarkets or online, for a total of ¥60,000 (about £300 or $400). Among those items are things like a towel warmer, a sports drink and a collagen-coated dish.

The idea is to replicate the kind of cell culture that happens in a lab. "When the right cells are placed in the right culture media, under the right conditions for a sustained period of time, cells proliferate," says Hanu.

Once you've bought everything you need, you take cells from a fertilised chicken egg, and you keep them at the correct temperature. That's what an incubator would be used for in a lab setting, but the DIY method substitutes that with a towel warmer.

The chicken cells also need something to stick to: a cell scaffold. That's what your collagen-coated dish is for. And you'll need to feed the cells with sugar, amino acids, vitamins and minerals – something Hanu calls the 'basal medium'. Hence, the sports drink.

> **"As of now, it may be better, but less excitingly, described as 'DIY cell culture'"**

ABOVE IntegriCulture's bioreactor can grow more than 30 types of cells from animals and fish **LEFT** An example of meat cultured in a lab

If you don't want to bother with all these items, you could just opt for IntegriCulture's cellular agriculture starter kit. It includes all the basal media, serums, starter cells and cell scaffolds you'll need, but will set you back at least £440 ($600).

Safety is a natural concern for anyone preparing food for consumption, especially when it comes to meat, due to the risk of food poisoning. But, according to Hanu, once you're up and running, the main priority is keeping your equipment clean. "Avoiding contamination, from things like mould and bacteria, is critical," he says.

Contamination can kill the growing cells and signs that they may have succumbed to it include the cells changing colour or floating in the basal medium, or the basal medium turning cloudy. None of which makes the process sound like it could result in a product that's especially appetising.

It's also worth pointing out that little is known about what potential for harm there is to humans in cultivated meat – from the lab or anywhere else – at the moment. It's reasonable to assume that you could accidentally culture something more dangerous than a small piece of chicken. So, if – despite the above – you're in any way tempted to try it, we'd advise against it for now.

QUANTITY AND QUALITY

Hanu says the Shojinmeat method can, so far, successfully produce about a gram of food, so not enough for a single nugget. Nevertheless, it serves as a proof of concept.

"We've made 'growing meat at home' a possibility, but the quantity and quality of the output needs thought," he says. "As of now, it may be better, but less excitingly, described as 'DIY cell culture'."

It's not just the amount of meat that needs improvement. In labs, cultivated meat can replicate complex textures, using plant fibres and 3D printers to imitate strands of flesh. But 'homegrown' meat hasn't quite reached this level.

"Producing a complete muscle tissue with fat marbling and texture involves advanced tissue engineering," says Hanu. "With our technology, the product is cultured cell mass, and the final product resembles Spam or sausage."

For now, Shojinmeat's work serves as a glimpse into what future food science could make possible – rather than an invitation to start cooking your own meat at home.

by **HATTY WILLMOTH**

Hatty is a trends editor at BBC Science Focus. *She has a background in health, nutrition and food.*

SHOJINMEAT, GETTY IMAGES

Finally…
An EV worthy of your bedroom wall

Ferrari's new Elettrica could be the car that gets dyed-in-the-wool petrolheads to long for an EV. It could also be the car that reshapes the entire EV landscape

by QUENTIN WILLSON

"Here's the new Ferrari EV." Five words I never imagined saying. And as I delivered my piece-to-camera at the famous Maranello factory in Modena, Italy, I felt a frisson of history being made. Because this is a very big deal.

The world's most admired automotive brand has been conspicuously absent for over a decade of battery-only EV industry transition. It just carried on doing what it's always done: building loud, wailing V12, V8 and V6 combustion engines. So, when the Prancing Horse suddenly changes strategic direction, the world sits up and takes notice. The announcement of the Elettrica is a defining moment in Ferrari's history.

A FERRARI, BUT ELECTRIC

For those at the back shouting 'Sacrilege! I'll never buy one!' Remember Enzo Ferrari's oft-repeated mantra: 'innovate, innovate, innovate.' As Ferrari's founder, he knew that pushing the brand's mechanical and technological envelope meant that his scarlet cars would win races and inhabit the posh, climate-controlled garages where serious money keeps its toys. Current CEO, Benedetto Vigna, told me there had been such major innovation in the Elettrica that "you have to show, step-by-step, what's in the car."

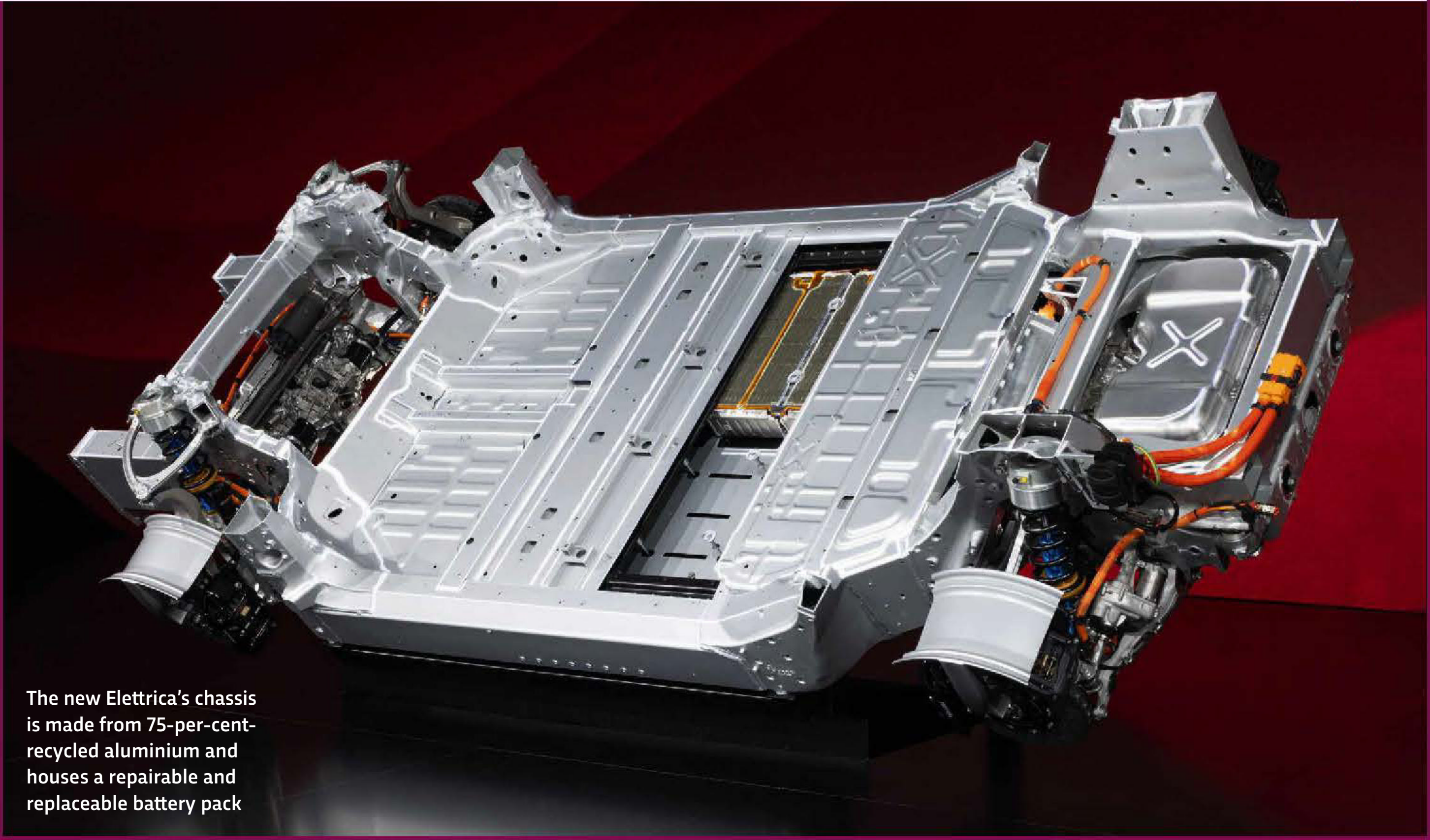
And while I was only shown the chassis, batteries, motors, axles and suspension – the complete car will be unveiled in 2026 – we do know it's a four-seater with four doors, like Ferrari's existing Purosangue, but won't be a supercar (current battery tech isn't reckoned to be good enough).

Four electric motors will provide 1,000 brake horsepower and a jumbo 122kWh battery, with 15 modules of lithium-ion pouch cells – one of the biggest in the industry – will keep the electrons stored and flowing with a charging speed of up to 350kW. For players of *Top Trumps*: 100km/h (62mph) comes up in 2.5 seconds; top speed is 310km/h (192mph) and battery range is around 530km (330 miles) from a weight of 2,300kg (5,000lbs).

Camouflaged prototypes have been doing night sorties along Moderna roads for two years and it was good to hear Vigna admitting to burning long midnight hours finessing that special Ferrari DNA on the hills around the factory. There's obvious passion for the project and no suggestion this is just a compliance car. Quite the opposite.

Vigna insists the Elettrica will perform at least as well as the company's internal combustion engine cars and will appeal to those who would – for whatever reasons – never normally drive a Ferrari. Product development chief, Gianmaria Fulgenzi, describes it as "an electric car, but first and foremost – conceptually and in practice – it is a Ferrari." He hinted at more versatility, visibility, comfort and practicality. A practical Ferrari for hands-on, eco-conscious, multi-millionaires.

The new Elettrica has yet to be fully revealed, but will be a four-door, four-seater like Ferrari's existing Purosangue



The new Elettrica's chassis is made from 75-per-cent-recycled aluminium and houses a repairable and replaceable battery pack

FERRARI X2

> “If battery chemistries change in the future, then the whole pack can be replaced with an entirely new chemistry”

FUTURE PROOF

With the heft of Ferrari's F1 expertise, we could see a process of EV development that no other car maker has been able to bankroll. EVs make very little profit (if any), so the leaders in battery and software development have been broadly Tesla and the Chinese.

Slowing markets have choked EV innovation (the new Tesla Model Y is no longer the gold standard) and car makers rely on sourcing batteries from Asia as well as outsourcing design and production of axles, suspensions, software and battery management systems (BMS) to third-party supply chains.

Ferrari has developed most of the Elettrica in-house. The BMS and battery pack are assembled on site and, critically, the pack is repairable and replaceable. This is a huge step forward in an industry that will often write off a £50,000 car for a minor dent in its battery pack casing.

The pouch cells are sourced from SK On in South Korea and are said to have “the highest energy density of any electric car on sale.” 20 fixing points anchor the pack to the 75-per-cent-recycled-aluminium chassis so it can be unbolted, opened up and any failed cells replaced.

But if battery chemistries change in the future, then the whole pack can be replaced with an entirely new chemistry. Even though data shows modern EV batteries are lasting well, Ferrari is taking no chances.

The same with battery cooling. A multiple flow circuit running between and beneath the cell pouches inside the pack is integrated into the car's primary cooling system. Inverters (the devices that convert and control the flow of electricity from the battery to the motors) have been engineered from scratch, there's active suspension and four magnet synchronous motors delivering 310kW at the rear and 105kW at the front. More engineering audacity has gone into the Elettrica than into any other EV before.

And what about the noise – that iconic V12 wail? Ferrari is horrified at the idea of having fake engine noise. Instead, a sensor on the rear axle will pick up the frequencies of the powertrain, which will then be amplified and projected like an electric guitar.

But the air-ship-sized question here, is will the Elettrica be a worthy bedroom wall poster car like all those Daytonas and Testarossas? I think Ferrari may surprise us. If you look at the engineering that went into its two hybrids – the SF90 and 296GTB – there's a level of innovation that we haven't seen anywhere else. The 296 hybrid was the fastest rear-wheel-drive Ferrari ever tested by *Car and Driver Magazine*. And, because of strong regenerative braking, my journey down the hills around the factory in a 296 generated almost as much electricity as I'd consumed going up. These are enormously clever cars.

This may only be stage one of a three-part reveal of the Ferrari Elettrica, but thus far, I'm impressed. But also, relieved that at last a proper car maker with proper resources could perhaps assume the technological leadership of the EV transition.

by **QUENTIN WILLSON**

Quentin is an award-winning motoring journalist and transport campaigner. He presented BBC Two's Top Gear *for a decade along with many other motoring and consumer shows.*

EDITOR'S PICKS...

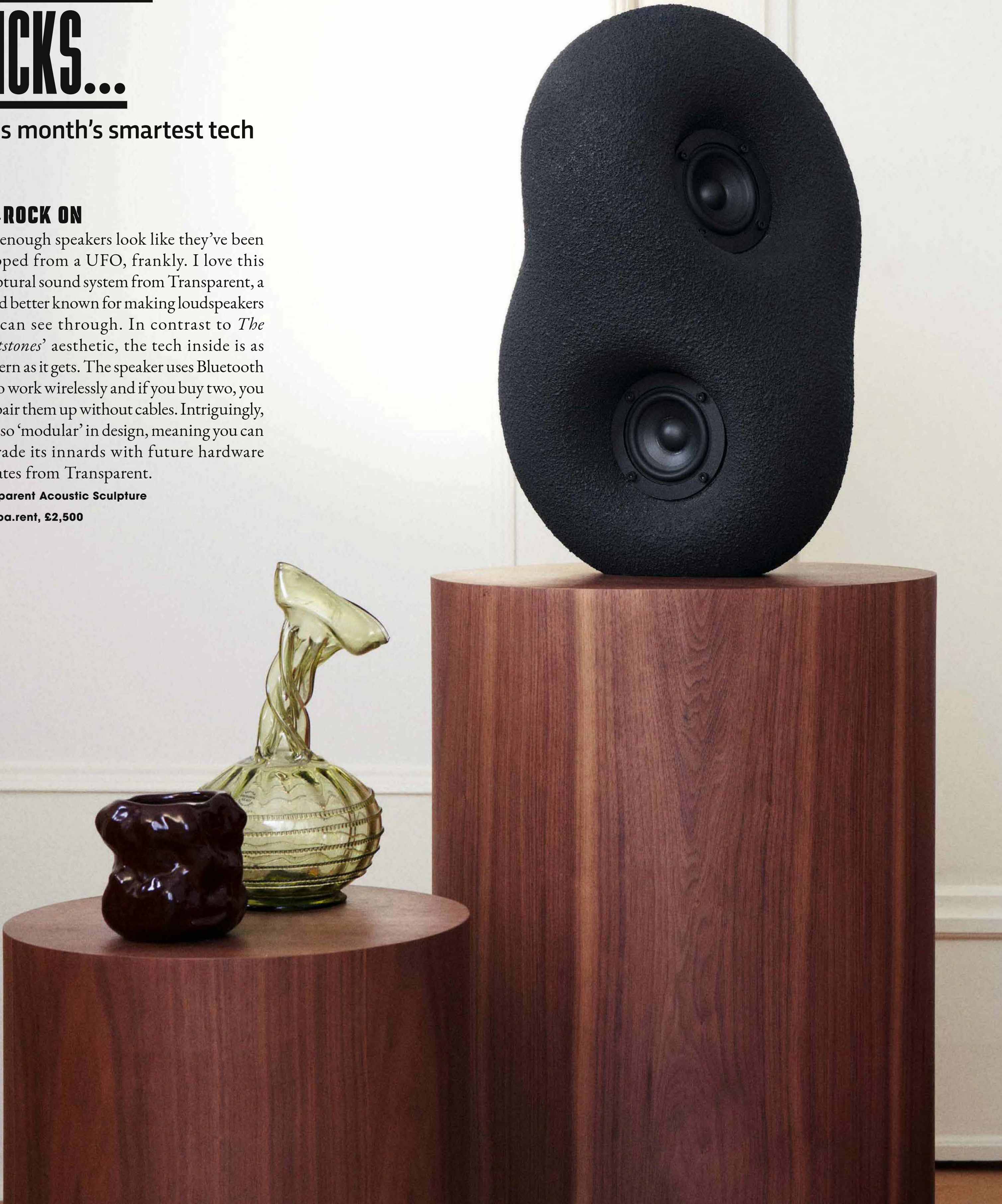
This month's smartest tech

→ ...ROCK ON

Not enough speakers look like they've been dropped from a UFO, frankly. I love this sculptural sound system from Transparent, a brand better known for making loudspeakers you can see through. In contrast to *The Flintstones*' aesthetic, the tech inside is as modern as it gets. The speaker uses Bluetooth 5.0 to work wirelessly and if you buy two, you can pair them up without cables. Intriguingly, it's also 'modular' in design, meaning you can upgrade its innards with future hardware updates from Transparent.

Transparent Acoustic Sculpture

transpa.rent, £2,500



↗ …LEFT, DOWN, UP, RIGHT

Another year, another edition of *EA Sports FC* (formerly *FIFA*) to remind me my gaming skills aren't what they used to be. If, like me, you're looking for an edge to prove to the next generation that you're not an 'unc', a pro controller might be worth your time. The G7 Pro does it all, it can be used wired or wirelessly and it works across Xbox, PC and Android. Clicky buttons give you tactile feedback when pressed, while the 'Hall effect' analogue sticks track movement by detecting changes in magnetic fields, rather than through the physical tracking used by standard controllers. This means there's less wear and tear, and no chance of physical drift (when your actual inputs don't quite match up with what's happening on screen).

GameSir G7 Pro

gamesir.com, £89.99

↓ …MADE OF STERNER STUFF

This could be the last backpack you'll ever buy. Described as an "Alpine Climbing System" by North Face, it's made from a fibre called Dyneema. The material's creators say it's the strongest fibre in the world, claiming it's 15 times stronger than steel by weight. This means that, as well as being incredibly tough, the backpack weighs less than a kilo (2.2lbs) when empty. Its features are modular, meaning you can clip on smaller holders and pockets as needed. The whole thing is waterproof, too, with taped seams designed to withstand downpours, rain and snow.

North Face Summit Advanced Mountain Kit Backpack

thenorthface.com, from £315

↗ …WOOD CHIP

Circuit boards are currently made from petroleum-based materials that can't be recycled – a real pickle when you consider the sheer number of electronics getting thrown out these days.

Enter a radical solution from researchers at EMPA: wood-based circuit boards. By mixing cellulose with lignin, a waste product from the paper-making process, the team has created a board that's fully biodegradable. Once the board itself has decomposed, the metallic and electronic components can be removed from the compost and recycled.

Though the boards remain sensitive to water and high humidity, the team plans to strengthen them without compromising on biodegradability.

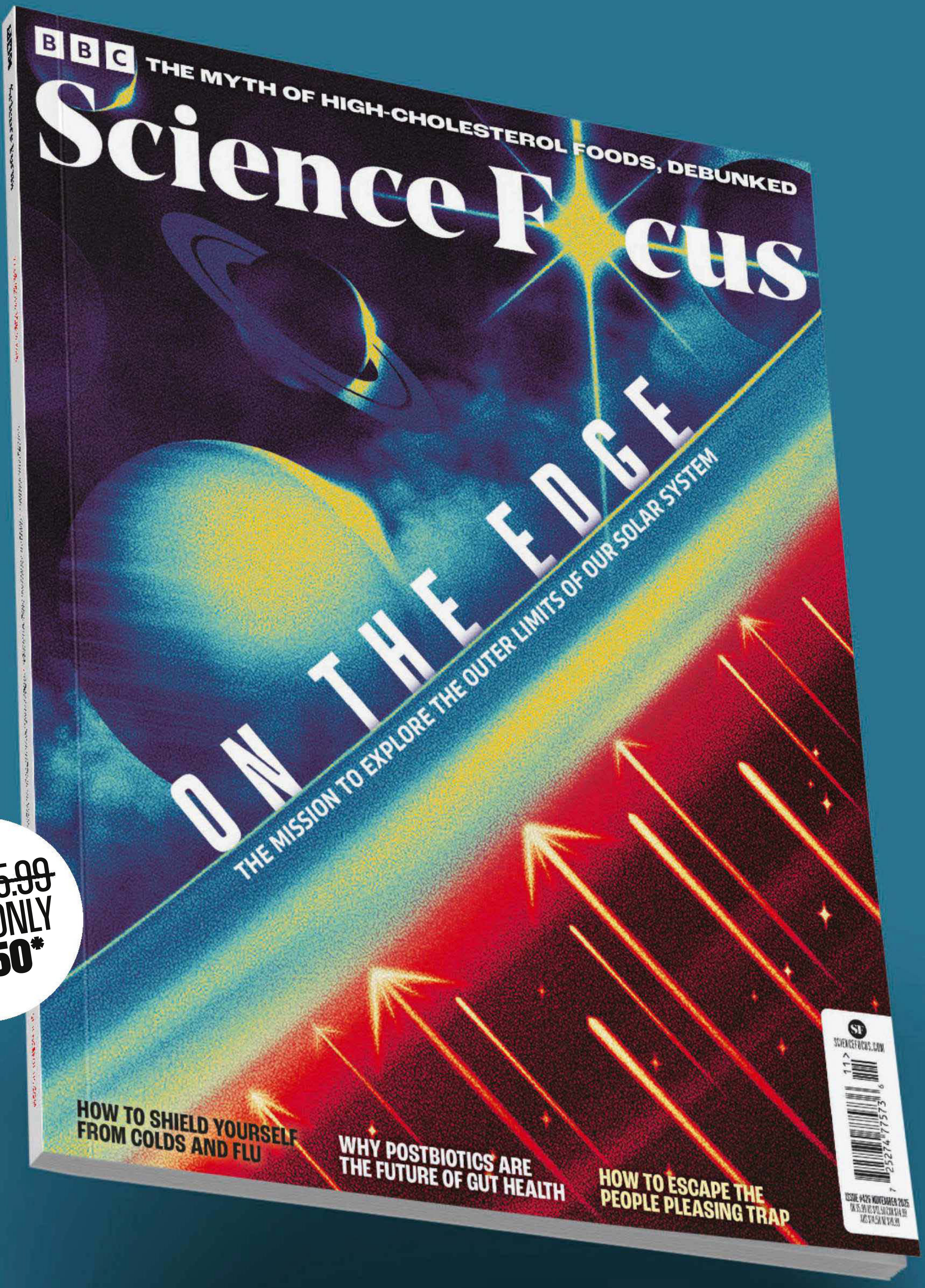
hypelignum.eu

↓ …REACH FOR THE STARS

These augmented reality-powered binoculars solve the biggest problem with binoculars: when you see something cool and pass them to a friend, they immediately lose sight of it. To fix that, Unistellar's Envision binoculars have a "target-lock" mode, so when you spot something interesting, you can hand them over and an on-lens overlay will guide your buddy straight to it. The same overlay can also identify and highlight mountains, lakes and rivers – as well as cosmic objects in the night sky.

Unistellar Envision

unistellar.com, £1,299



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IMMUNE TO

GETTY IMAGES X2

EVERYTHING

Researchers are edging closer to a universal antiviral drug, a single medicine that could – if it works – treat every virus known to man and head off the next pandemic before it even starts

by **IAN TAYLOR**

Imagine coasting through flu season with barely a sniffle. Or brushing off COVID, no matter how many times it mutated. Imagine, in fact, that no virus can harm you, from chickenpox to Dengue to HIV. Even the deadliest viruses we know of, like rabies or Ebola, don't cause you serious problems.

For a handful of people, this seems to be the case. Anyone with a specific and rare genetic mutation benefits from a superpowered side-effect: they fight off viruses with ease, to the extent that most of the time, they don't even know they've been infected.

The mutation in question causes a deficiency in a key immune system protein called ISG15. In turn, this leads to a mildly elevated systemic inflammation in their bodies – it's this inflammation that seems to subdue any virus that tries to get past.

When Dusan Bogunovic, professor of immunogenetics at Columbia University in New York, first discovered the mutation 15 years ago, he didn't realise what was in front of him.

"I don't know that 'ignorant' is the right word, but I was unaware of this possibility for the first three years after we discovered it," he says. "I wasn't studying viruses."

It was only when he took a job at a lab that was studying viruses that it dawned on him. He remembers going to seminars to learn more.

"That was the 'Eureka moment'," he recalls. "That was when I suddenly thought: 'Wait, the autoinflammation profile in these rare patients is 100-per-cent antiviral!'"

Sure enough, when he went back from the seminars and studied the ISG15 patients' immune cells, Bogunovic found they had encountered several viruses during their lives, including flu, measles, mumps and

From fogging and vaccines to potentially universal antivirals, the more ways we have to tackle virus outbreaks, the more options we have to protect people

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chickenpox. Only these patients had never shown any of the symptoms associated with them. Their immune systems, in a permanent state of high alert due to their genetic mutation, had never allowed the viruses any kind of foothold.

"Think about when someone in your household gets the flu," Bogunovic says. "They're floored by it, but the person they share a bed with only gets a little bit of sniffles and a sore throat. In a way, you can think of this mutation like those people who just go through the sniffle phase.

"What's even cooler is, it's your own DNA that makes you ready when the virus comes. The machinery that's evolved to fight off viral infections is just slightly enhanced."

The discovery gave Bogunovic an idea. A big one. What if we could create a drug that mimics the effect of the mutation? And what if you could control the inflammation response so that your levels aren't chronically elevated, like people with the mutation? You just switch it on when you need it – when there's a virus.

The possibilities for such a drug didn't end there, however. What if it didn't treat just one virus, but entire viral families? In fact, what if you could make a universal antiviral that was effective against every virus we know of, and even the ones we don't? Such a drug would be world-changing – a medical multitool unlike anything we've seen before in the treatment of viral infections.

It wouldn't just mean we all feel better in the winter. This kind of breakthrough would save countless lives and massively lower the burden on public health systems worldwide. Then, if another pandemic-level virus, like COVID, emerged tomorrow, we'd have a treatment on the shelf that could stop it in its tracks.

MIRACLE MEDICINE

A single drug that's effective against every sort of virus sounds like a researcher's idle daydream, the ultimate medical what-if. The thing is, Bogunovic and his colleagues may have already achieved it, or at least something close.

Last summer, they published results of an experiment in which mice and hamsters were given a drug inspired by people with ISG15 deficiency. It's a cocktail of 10 gene products, all of which are elevated in people with the mutation. Researchers exposed the animals to influenza and SARS-CoV-2, the virus that causes COVID. Neither virus was able to replicate in the animals, nor cause them severe symptoms.

"This was the first time in vivo – in living animals – showing that this drug can work," Bogunovic says. "Of course, prior to that, we tested it against 10 different classes of viruses in vitro and couldn't find one against which it didn't work."

In other words, in cell cultures in the lab, no virus has been able to break through the treatment's defences.

Broad-spectrum antivirals – drugs that treat multiple viruses – are not a new idea. Remdesivir, for example, is an antiviral used to treat COVID, but it was originally developed for hepatitis C and has also been tested for Ebola, albeit with limited results.

> "WHAT IF YOU COULD MAKE A UNIVERSAL ANTIVIRAL THAT WAS EFFECTIVE AGAINST EVERY VIRUS WE KNOW OF, AND EVEN THE ONES WE DON'T?"

Research into broad-spectrum antivirals has intensified over the last 15 years in response to multiple viral threats such as Middle East respiratory syndrome (MERS), Zika virus, Ebola and monkeypox. And that's to say nothing of COVID, the biggest health crisis in a century, which unsurprisingly renewed interest and fast-tracked research money (including a large effort to repurpose existing drugs for the new viral threat).

But broad-spectrum antivirals are difficult to develop, mostly because drugs that treat viruses usually target proteins on a virus's surface. For a drug to work on multiple pathogens, the target protein would need to be the same from virus to virus. This is rarely the case, even in closely related viruses. There are drugs that work for SARS-CoV-1, for example, which don't work for SARS-CoV-2.

Even so, it seems that research is now paying off because Bogunovic's team is not the only one to report impressive results. In 2025 alone, multiple groups have published research on broad-spectrum antivirals.

SUGARCOATED

One particularly promising and novel option comes from a team of researchers led by Prof Adam Braunschweig at the City University of New York and Prof Hector Aguilar at UCLA. Together, they have created a drug that targets sugars on the surface of a virus rather than its proteins. While proteins are different from one virus to the next, sugars are largely the same.

"That's because it turns out viruses don't make sugars," says Braunschweig. "They steal them from the host. So in fact, most of the COVID circling around in your body is covered with sugars that it stole from you. And if these sugars are made of you, your body doesn't know that they're bad. That's how the virus can enter the cell."

The team's new therapy binds to these same sugars. It stops viruses from evading the host's immune system and invading the cells. In lab tests, Braunschweig and his colleagues have tested their drugs against multiple viruses, representing five viral families. In a smaller study in mice, a single treatment protected 90 per cent of animals against COVID.

"It's effective against 100 per cent of the viruses we've tested it against," Braunschweig says. "These include influenza viruses A and B, bird flu and filoviruses, like Ebola and Marburg."

Braunschweig and his colleagues are already planning human trials, currently scheduled to begin in two years. Meanwhile, other researchers continue to explore different ways of developing broad-spectrum antivirals.

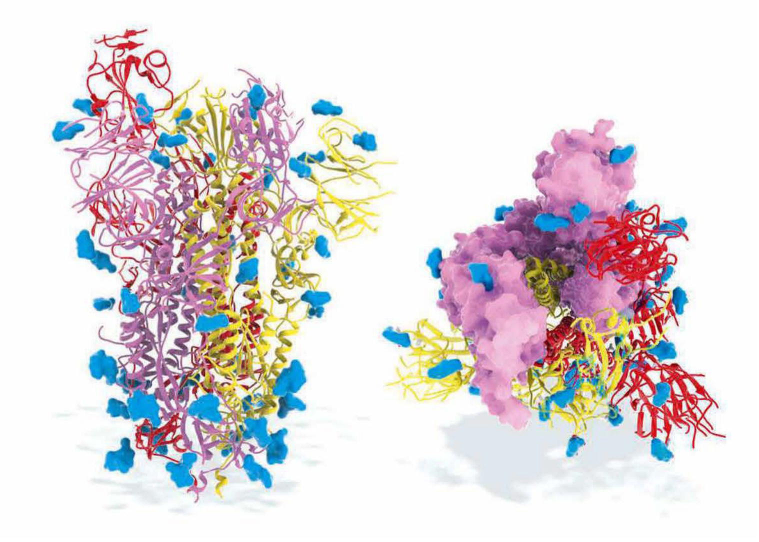
A team in Germany found that a drug that treats diabetes could also cut the viral load of viruses like SARS-CoV-2 and Dengue. Other researchers, forming a global non-profit collective called the COVID Moonshot, have developed an open-science broad-spectrum drug candidate for coronaviruses. Protease inhibitors are yet another option, with drugs targeting enzymes that viruses need to make copies of themselves.

The race is very much on to find a universal antiviral, but there are also voices urging caution when it comes to the research,

A universal antiviral may have prevented the need for lockdowns during the COVID pandemic including scientists who have already made broad-spectrum drugs.

"I'm sort of a poster child for this," says Prof Raymond Schinazi, a virologist at Emory University in Atlanta, Georgia, "because I discovered drugs for HIV that also work against hepatitis B." He thought it was a quite the breakthrough until he realised the potential for both viruses to mutate and develop a resistance to those drugs. Or the other alternative: that by treating one condition you get resistance to the other. "Then you're in trouble," he says, "real trouble."

Schinazi says two camps are emerging among scientists: those who are excited about the potential of broad-spectrum drugs and those who prefer very specific compounds. He belongs to the latter.



LEFT Computer models showing how SARS coronavirus proteins use the host's sugars (blue) to disguise themselves and hide from the immune system

BELOW A universal antiviral may provide added protection for frontline health workers during outbreaks

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> **"THE QUESTION IS WHETHER YOU CAN FIND A THERAPEUTIC DOSE THAT'S FAR FROM A TOXIC DOSE"**

"We are very keen, in my lab, on finding a specific antiviral agent. One virus, one antiviral," he says.

His point is not that silver bullet antivirals won't work, it's that we need back-ups: multiple drugs that work in multiple ways, combined with vaccines, to give several layers of protection against a given virus.

"It's a bit like driving a car," Schinazi says. "You need the seatbelt and you need the airbag. Same thing with viruses. If you have vaccines available, as well as drugs, you're beautifully set up in case a virus blows up. You're ready to annihilate that virus."

UNINTENDED CONSEQUENCES

There are other challenges facing the researchers who are developing these treatments. One is the risk that a compound is so wide-acting that it does something you don't want it to do. "We are always worried about side effects and off-target effects," says Schinazi.

It's something Braunschweig is keenly aware of in his research. The type of sugar his team is targeting doesn't just appear on the surface of viruses; it appears on every human cell. "We're targeting something that we obviously need," he says.

So how do you make sure the drug you're making isn't toxic to the people you're trying to protect?

"Well, all drugs have toxicity at certain concentrations," Braunschweig says, from cancer drugs to paracetamol. "I think Tylenol [paracetamol] has probably the most unintended overdoses for an over-the-counter drug because people take huge dosages.

"The question is whether you can find a therapeutic dose for your drug that's far from the toxic dose. It's whether you can target something with an acceptable therapeutic window. For me, the upside is so big that it was worth trying."

The compound Braunschweig and his colleagues have developed seems to have a very broad window. In other words, at this preclinical stage, it's safe.

"We see some toxicity at 100 milligrams per kilogram, [mg/kg]" he says. "But the therapeutic dose that we tested was 2mg/kg. We also tested 10mg/kg and observed no toxicity whatsoever."

The next challenges for researchers are practical, logistical and financial. Braunschweig and Bogunovic are both looking ahead to human trials. But first, there's a molecular engineering job to be done to ensure the drugs are packaged and delivered in a way that works safely.

"That technology is improving, but it's not perfect yet," Bogunovic says. "Then you have the fact that this is a new class of drugs against infections. So you need partners in regulatory bodies who understand and want to advance this in the most ethical, standardised, appropriate way. And then, of course, somebody has to finance it."

Money could be a serious barrier. For all the research grants awarded after the COVID pandemic, clinical trials are still notoriously expensive.

"From drug optimisation to phase one [the first time a new drug is tested in humans], and then phase one through to a drug that's approved by regulators, we're probably talking about a billion dollars," Bogunovic says. "And how do you run a clinical trial for a universal antiviral? You have to run it virus by virus, which makes it extremely expensive."

For biotech companies that need a return on their investments, it's high-risk, especially when it comes to preparing for the next pandemic. Because in that scenario, you're effectively developing drugs that we all hope we never need.

"If I told you, the investor, there's going to be SARS-CoV-3 in two years, but we can get a drug to the market in time," Bogunovic says, "that's great. We help people, people won't die and we make a profit.

"But I can't tell you what will happen. In fact, I hope it doesn't happen."

So how do you convince investors to fund the drug anyway? Bogunovic believes we need public-private partnerships, where governments fund and stockpile broad-spectrum drugs. If we'd done that before 2020, the COVID pandemic would have looked very different, he says. "It's hard to say for a fact, but if we'd had these drugs in 2020, I do think we would have saved lives, we would have saved the economy, and we would have saved mental health and education."

PANDEMIC PREPARATION

The threat of another viral pandemic has hardly gone away. Governments and researchers alike have repeatedly stated the need to learn the lessons from COVID. But when life returns to normal, and when everything from wars to public services to climate change is competing for public money, it's easy to collectively forget that viruses are one of the biggest threats to humanity's continued survival.

ABOVE Health services were under great pressure during the COVID pandemic. A universal antiviral could reduce the number of people requiring hospital treatment during a potential future pandemic

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"WE CAN IMAGINE A MOLECULE THAT'S A DOLLAR A DOSE AND WORKS AGAINST EVERY VIRUS"

That's what makes a universal antiviral so appealing. A drug with far-reaching effects could lessen the burden of viruses that exist today and – hopefully – work for the next one that emerges.

In the pandemic scenario, Braunschweig says the biggest impact would be to prevent the infected from becoming patients and save health services from being overwhelmed like they were in 2020. "Let's say this stuff works like broad-spectrum antibiotics. You may still suffer, but it lowers the intensity of the disease. Then only the most severe cases need those hospital beds."

And in the meantime, these new drugs could give us the upper hand in the perennial fight against viruses.

"It's hard to overstate the potential impact," Braunschweig says. "We can imagine a molecule that costs a dollar a dose and works against every virus with at least some impact. The spectrum could be from HIV to the common cold, to Nipah and Hendra. In other words, it could eliminate as a threat our greatest enemy throughout the history of our species." **SF**

by **IAN TAYLOR**

Ian is a freelance science writer and the former deputy editor of BBC Science Focus.

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WHAT WE GET WRONG ABOUT COLD AND FLU SEASON

Nobody enjoys being stuck in bed sneezing and coughing the days away. But there are steps you can take to increase your chances of avoiding these winter ailments

By **Holly McHugh**

As the winter months draw in, so does the yearly wave of colds and flu, making now the perfect time to think about protecting yourself and others. You know the drill: wash your hands often, cover your coughs and sneezes, avoid touching your face, keep indoor spaces ventilated, stay home if you feel unwell and get a flu shot.

But beyond this familiar advice, there are also useful steps you can take that you might not know about. Here are the latest findings and lesser-known strategies to help get you and your family through the cold and flu season.

HUMIDITY IS KEY

It's common knowledge that ventilation is important to help stop the spread of cold and flu viruses. But what's often overlooked and plays an equally important role in how these viruses spread is humidity.

Research shows that flu viruses survive longer in dry air, which is why outbreaks often peak in winter when central heating dries out our homes and workplaces. Low humidity also makes the tiny droplets we exhale evaporate into even smaller particles. These can float in the air for longer and travel further, giving the virus more chances to spread.

Dry air also weakens the body's natural defences by drying out the protective mucus in our eyes, nose and throat. This makes it harder to trap and clear away germs, which not only increases the risk of infection, but also slows recovery time and makes symptoms like coughs, sore throats and congestion worse.

Evidence suggests that maintaining indoor humidity in the 'Goldilocks zone' of 40–60 per cent makes the environment less hospitable to viruses, while helping our airways stay moist and better able to fight off infections.

Although it's important to keep crowded spaces ventilated, in winter it often brings in cold, dry air that actually lowers indoor humidity. That's why scientists recommend balancing fresh air with the right level of humidity to help reduce the spread of colds and flu.

The most effective way to add moisture to the air is with a humidifier, especially those with built-in controls that regulate humidity levels.

THE EXERCISE MYTH

For decades, the conventional thinking was that endurance sports temporarily suppress your immune system, increasing the chances of illness. But in 2018, Dr John Campbell, senior lecturer in the department of health at the University of Bath, turned this idea on its head. His research showed that exercise could actually boost the body's immunity.

"When we do strenuous exercise, the number of immune cells circulating in the blood temporarily increases. After exercise, levels drop below baseline, which was once thought to be an 'open window' for infection," says Campbell. "We now know that these immune cells aren't lost. Instead, they move into tissues that are more likely to become infected, such as the lungs, to hunt for infections. This suggests that exercise can actually enhance the immune system."

Campbell stresses that exercise is good for health and helps protect against disease, so people shouldn't fear that it weakens immunity. He says, "Aerobic exercise, such as running or brisk walking, is great for boosting the circulation of immune cells. We also now know that resistance training (lifting weights) plays a role too, as muscle tissue releases proteins that support immune function. Combining both types should give the best, broadest support to your immune system."

> "AEROBIC EXERCISE IS GREAT FOR BOOSTING THE CIRCULATION OF IMMUNE CELLS"

Vitamin D supplements, particularly D3, plays a vital role in supporting immune health during the winter

PRIORITISE VITAMIN D OVER VITAMIN C

While vitamin C may help to slightly shorten the duration of an illness, there's unfortunately little evidence that it can prevent us from getting ill in the first place. Vitamin D, on the other hand, has been proven to help strengthen our immune systems.

18 per cent of adults aged 19–64 in the UK are deficient in vitamin D. The NHS recommends that everyone take a daily supplement of 10 micrograms of vitamin D during the darker months (October to March).

There are two types of vitamin D supplements available: vitamin D2 and vitamin D3. A recent study,

however, found that taking vitamin D2 may actually lower our levels of vitamin D3 – the kind our bodies use to support the immune system. “We found that people given vitamin D2 had a drop in vitamin D3 levels, even compared with those who took no supplements at all,” says Emily Brown, lead researcher of the study at the University of Surrey.

Prof Susan Lanham-New, co-author of the study, adds: “Vitamin D2 seems to activate an enzyme in the body that speeds up the breakdown of vitamin D3. Studies also show that D3 switches on helpful genes that strengthen antiviral and antibacterial defences, while D2 doesn’t appear to have this same effect.”

Brown stresses that everyone in the UK should still take a vitamin D supplement: “Our study suggests that vitamin D3 is the best option, but further research is needed to fully understand the long-term differences.” **SF**

ALAMY X2, GETTY IMAGES

by **HOLLY MCHUGH**

Holly is a freelance science writer who specialises in health.

FLU JABS AREN’T JUST FOR GRANDPARENTS

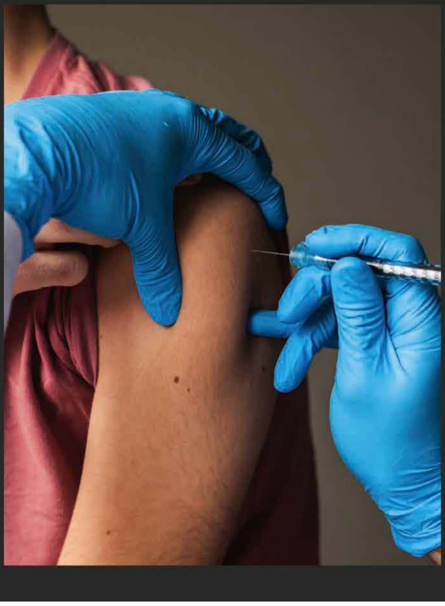
Another common myth is that only older people or those with serious health conditions need the flu jab. In reality, flu can affect anyone. Even healthy adults can become seriously ill.

Being essentially little germ factories, children are the biggest spreaders of flu. It can also make them very unwell because their immune systems are still developing, and their smaller airways make them more prone to serious complications such as bronchitis and pneumonia. In fact, children under five have some of the highest rates of flu-related hospital admissions.

It’s for these reasons that the UK government recommends the flu vaccine for all children aged two and above, up to the age of 16.

This year, it expanded the vaccination programme so that two- and three-year-olds can also get their jab at community pharmacies, making it easier for families to protect their little ones, as well as everyone around them.

Many people also don’t realise that, as an adult, you can still get a flu jab privately, even if you’re not eligible for a free NHS one. Pharmacies and some GP practices offer it for a small fee, usually at a price point of around £15–£25, so you can cheaply and efficiently protect yourself if you’re worried about catching flu.



THE PEOPLE PLEASING TRAP

(AND HOW TO ESCAPE IT)

Are you a doormat if you constantly make sacrifices for other people? Or are you simply being a nice person by putting others' needs ahead of your own? Psychologists think there's something deep at work

by **AMY ARTHUR**

ILLUSTRATION: ALICIA BLASCO

Growing up, I spent a lot of time with my grandparents, who instilled in me the importance of politeness and the authority of adults. These principles turned me into the kind of child that teachers loved – and that my peers mocked.

I raised my hand to answer every question and volunteered to hand out books at every opportunity. It could've been that I was innocently trying to be a good student, but I'm sure I also thought that being in my teachers' good books might give me some kind of academic advantage. I was people pleasing.

As I grew up, I found new people to please. Lecturers, recruiters, strangers on the internet. I hid behind the excuse that I was saying 'yes' to things I really wanted to say 'no' to, because it would further my career. And by doing so, I was really saying 'no' to things that could've offered me a happier life, like spending time with family.

But I reasoned, that as someone at the start of what I hoped would be a long, successful career, what was wrong with a little personal sacrifice?

"There are many reasons why we do nice things for other people," says Prof Toru Sato, a psychologist at Shippensburg University in Pennsylvania, in the US. Sato began studying people pleasing in the early 2000s, concerned with its link to depression and anxiety.

One reason people pleasers make sacrifices is in the hopes of receiving something later down the line. "We do that all the time, right?" says Sato. "We do it at work and in relationships with people: 'I scratch your back, you scratch mine'."

If everyone in the transaction feels happy with what they give and what they receive, the process is relatively neutral (and not harmful). For example, a people pleaser at work who puts in extra hours in the hopes of receiving a promotion may be happy to do so, provided their organisation rewards them.

Another reason is genuine care for a person. "If your child is sick and you're doing what you can to help them heal, you're not doing it because you expect some kind of reward. You just genuinely care about their wellbeing," says Sato.

This might be what a people pleaser believes they're doing when they sacrifice their priorities and needs for someone else's agenda, but according to Sato, there's a hidden motive operating beneath these behaviours.

The reason that true people pleasers make sacrifices for other people isn't for a reward, or out of kindness. "They're actually trying to feel better about themselves," Sato says. "They feel like they're not good enough and so, in order for them to feel valued, they think, 'I need other people to like me, to respect me, to care about me'."

THE NEED FOR APPROVAL

Sato, along with other psychologists studying people pleasing in the early 2000s, was interested in the behaviour as part of a personality trait known as sociotropy. This trait is used to refer to the tendency to place excessive value on relationships and has been linked to higher rates of depression.

People who have a strong sociotropic element in their personality will also seek approval from others, be over-concerned with what other people think of them and, importantly, will rely on others' opinions to boost their self-esteem, says Sato.

Like other personality traits, sociotropy seems to exist on a spectrum. All of us will, in one way or another, care about what other people think and seek social approval.

> "ALL OF US WILL, IN ONE WAY OR ANOTHER, CARE ABOUT WHAT OTHER PEOPLE THINK"

Some psychologists have gone so far as to say that the desire for strong and stable attachments with others is a "fundamental human motivation", and this has likely been reinforced throughout human history.

When the earliest human groups formed to share resources and shelter their members from harsh environments, keeping a place within a community was a matter of survival. When social exile could mean a death sentence, developing strong connections and proving you could make worthy contributions to a collective was crucial. The resulting social approval for such contributions then encouraged positive behaviours to be repeated, benefiting everyone in the group in the long run, psychologists say.

The issue, according to Sato, is that some people with high sociotropy will place almost all their self-worth in the hands of others, seeking approval because it's the only way they know they're valued.

"They'll think things like, 'I don't feel like I'm good enough, that I'm worthy of being loved and respected and cared for if I don't please other people or if other people don't like me'," he says.

Sato's work has confirmed what others have found: sociotropic people are more vulnerable to depression and anxiety. "The theory is that if [your attempt at people pleasing] doesn't work out – meaning the other person didn't respond favourably to you – then you're vulnerable to being deeply affected, because you place so much emphasis on what other people think about you," says Sato.

ILLUSTRATIONS: ALICIA BLASCO

Someone with a healthy level of sociotropy might be able to handle a friend getting annoyed at them, for example, but an intensely sociotropic person would feel that their entire self-worth was under threat.

Being highly sociotropic has also been shown to make a person more submissive and exploitable. Their people pleasing puts them at risk of being taken advantage of.

Psychologists don't yet know why some people are more sociotropic than others, but research suggests that the trait appears alongside other personality characteristics like neuroticism and agreeableness.

Gender also plays a role in this spectrum, with women tending to score higher on sociotropic personality questionnaires than men.

One study found that women with high levels of sociotropy were more likely to experience stress in their relationships. The women could make their partners feel guilty, for example, by sacrificing friendships to prioritise their romantic relationship, or the women's reliance on approval made their friends feel pressured to dole out praise. These stressful encounters led the women to experience an increase in depressive symptoms, such as prolonged feelings of worthlessness or regret.

Living with high sociotropy is stressful. And though a sociotropic person will strive to satisfy everyone they have a relationship with, research suggests they're likely to cause their close friends and family more pain than pleasure.

THE MEANING OF SACRIFICE

In 2007, Sato and a colleague interviewed 261 university students to find out how they acted towards strangers, close friends and family members. They found that those with higher sociotropic personalities would be welcoming and friendly when they were in the presence of strangers, but could then be controlling, calculating and even vindictive to the people they were closest to.

This sounds counterintuitive to the nature of people pleasing, but Sato believes that as sociotropic people want to be liked by everyone, they'll put a lot more effort into establishing a good relationship than keeping one. When sociotropic people are already close to someone, they may feel "that a high level of relatedness can be maintained even without warmth and nurturance," Sato wrote in his paper.

He's also theorised that a highly sociotropic person might believe that once they've reached a certain quality of relationship, the only way to maintain it might be to carefully manage and control every interaction. While the sociotropic personality trait describes the people pleaser who will go to great lengths to receive approval from everyone, there are also those who seem to take on the responsibility of pleasing a select group of others, such as parents or a spouse.

Dr Mariko Visserman, a social psychologist at the University of Sussex in the UK, has spent her career studying self-sacrifices within close relationships, typically between romantic partners. She says that it's in these relationships where there tends to be the most tension between what two people want, and where making sacrifices can have a big impact on the way people feel about themselves and each other.

Visserman's work has looked at a wide range of sacrifices that people can make for the ones they love. This ranges from the seemingly small – like a person agreeing to have sushi for dinner when they really want pizza – to big life factors, like moving to a new city to benefit a partner's career, or whether to have children together. Her research suggests it's not the size of the sacrifice that matters, but the reason why it was made.

"If the decision is really about trying to benefit a partner because you care about them, then that's a very positive motivation for making a sacrifice," says Visserman.

This is what's known in psychology as an approach motivation, because it involves trying to approach, or bring about, a positive goal with a sacrifice. The opposite of this is

HOW TO LIMIT YOUR PEOPLE PLEASING BEHAVIOUR

1. Find your value internally, rather than in others' opinions

Explore where your sense of self-worth comes from. Research suggests we benefit from finding value internally, through identifying goals and values to live by, rather than seeking external approval or achievement. Maybe you pride yourself on how patient you can be with others, or value your pursuit of knowledge.

2. Practise better awareness of your thoughts

Becoming more mindful of your thoughts will help in several ways. According to Prof Toru Sato, a psychologist at Shippensburg University, mindfulness can help stop people pleasers from automatically saying 'yes' when they really want to say 'no', by allowing their real feelings to surface and balance the need to please.

This awareness can also help boost your self-worth: once you've identified things that make you feel good about yourself, you can try daily self-affirmations. This involves internally highlighting any acts that align with these values.

3. Break the habit of saying 'yes'

It can be difficult to stop yourself from people pleasing if it's become habitual. Instead, we can use lessons from the psychology of decision-making to break a habit and replace it with a new one.

First, identify the trigger of the habit. Is it an environment, like in the workplace? Or is the trigger a particular person? Then, plan in detail how you want to respond to that trigger. The more specific, the better – come up with a list of phrases that you can use to politely but firmly turn people down.

4. Be open to conversation

As Dr Mariko Visserman, a social psychologist at the University of Sussex, explains, our perception of sacrifice depends on how the recipient reacts. If you feel that the most painful part of your people pleasing is a lack of appreciation, you could invite your partner or friend to discuss how future conflicts might be settled in a way that makes both perspectives feel valued.

an avoidance motivation, in which you might be people pleasing because you're trying to avoid a conflict.

"This is where you might sacrifice for them because you want to avoid getting into some sort of tension or argument, or you want to avoid them being upset... or to avoid feeling like you're not a good partner," says Visserman. "If you see people pleasing as avoiding upsetting a person, then that would be more like an avoidance motive and that wouldn't be very helpful."

In fact, Visserman says that her research consistently shows that when a person's decision to sacrifice is motivated by a positive outcome, the result is beneficial for the relationship and for both parties in it. These sacrifices can actually be good for the couple.

"But if you make sacrifices and do things in your relationship to avoid negative outcomes or conflict, then that's actually related to the negative outcomes that you're trying to prevent," she says.

Whether either later on or further down the line, these kinds of sacrifices lead to conflict and less happiness between partners.

With either motivation, however, over time, excessive sacrifice can harm both parties. "If your partner is making a lot of sacrifices for you, that's not necessarily a good thing," says Visserman.

"Research has shown that people have mixed feelings when a sacrifice is made for them. They can feel grateful and hopefully they express that – which is good for the person making the sacrifice – but then, at the same time, [they can] also feel indebted. They might feel pressure to repay that sacrifice, or they might feel guilty that they didn't [make a] sacrifice."

As with sociotropic behaviours, people pleasing might not actually result in anyone feeling pleased. Instead, it can feel like a burden for the person on the receiving end of these sacrifices, Visserman explains.

YOUR SENSE OF SELF-WORTH

People pleasing, then, is not just about what one person does (or doesn't) do for another. It's also dependent on the behaviour of the person the sacrifice is being made for.

"We've done experiments presenting participants with a scenario of the exact same sacrifice and then manipulating how their partner responds.

"If it feels like their partner doesn't really understand what is important to them, or doesn't really care about it, then they would really regret the sacrifice and feel like it's a waste," says Visserman.

But, when we do feel like a partner is grateful, when they acknowledge what they sacrificed, the whole experience is much more positive. If a sacrifice seems to be appreciated and valued, it might not feel like people pleasing at all.

Both Sato and Visserman say that to reduce people-pleasing behaviour, you first have to understand why you feel the urge to please others.

If you believe your value or worth in life is based on the strength of your relationships, this is what psychologists call a 'contingent self-worth'. This means your self-worth is unstable: as soon as one of your relationships is threatened, it shakes your whole sense of self.

Our self-worth usually comes from several sources, however. One commonly used scale to measure self-worth identifies seven domains: approval from others, physical appearance, competition with others, academic achievement, familial love and support, alignment with personal values and morals, and spirituality or religion.

> **"CONSIDER HOW YOU CAN TURN EXTERNAL SOURCES OF VALUE INTO INTERNAL ONES"**

We don't need to feel value in all seven domains, it should be noted. Instead, most research points to an unhealthy self-worth being one based on purely external sources (like the approval of others), while a relatively healthy self-worth is based on internal sources.

So, if you want to boost your self-worth, you might consider how you can turn external sources of value into internal ones. For example, the thought that 'I am only a good person if other people like me' could become 'I feel good when I do things for the people I love'.

With the latter outlook, any sacrifices we make will be to approach a positive outcome, rather than to avoid a negative one. And all of us will need to make sacrifices throughout life, as Visserman's research has shown. We'll also have people make sacrifices for us, sometimes without us even realising.

What matters, then, is not that we should always say 'no' when the opportunity to people please arises. Instead, we need to feel secure in our ability to decide what's best for us and our relationships at that juncture.

If we then decide to please another person, we'll likely end up pleased ourselves. **SF**

by **AMY ARTHUR**

Amy is an award-winning writer, science journalist and author of Pace Yourself: How to have energy in an exhausting world (Black & White Publishing, 2023).

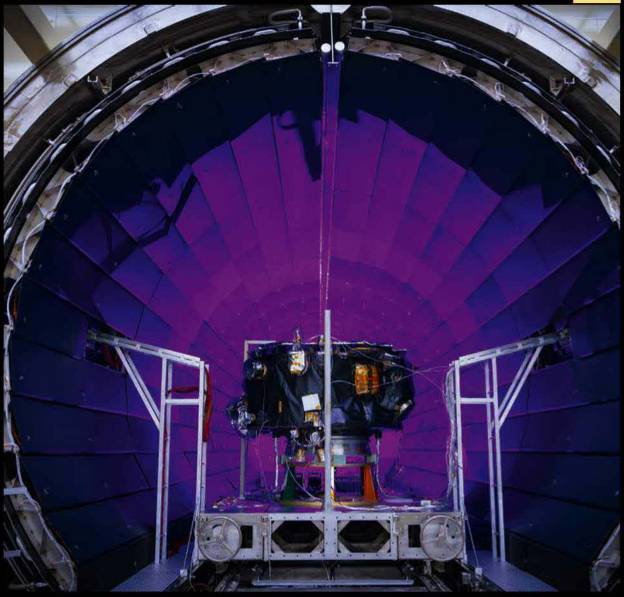
ILLUSTRATIONS: ALICIA BLASCO

THE QUEST TO FIND THE EDGE OF THE SOLAR SYSTEM

NASA's newly launched IMAP mission is set to tell us more about the boundary between our Solar System and interstellar space than ever before

by **JONATHAN O'CALLAGHAN**

ILLUSTRATION: MAGIC TORCH



ABOVE The IMAP was tested in NASA's thermal vacuum chamber to simulate the harsh conditions of space

Earth exists in a bubble. Our atmosphere forms a protective barrier between everything on the planet's surface and the near-empty vastness of space. But it's not the only bubble Earth sits inside. Beyond our familiar atmospheric cocoon lies a much larger bubble, an invisible boundary carved by the Sun itself.

This bubble, known as the heliosphere, is enormous. It encompasses the entire Solar System, spanning such a vast distance that only two spacecraft have ever managed to leave it. Launched in 1977, NASA's Voyager 1 and 2 passed beyond the heliosphere into interstellar space – the region between stars – in 2012 and 2018 respectively. They're the first man-made objects to ever travel beyond the Sun's protective bubble, and are still transmitting data about the charged particles and plasma waves that can be found there.

But astronomers want to learn more about the heliosphere and what lies beyond it. What's its exact size and shape? How do solar particles emitted by the Sun interact with interstellar space once they pass through it? And how effective is it at protecting us from the high-energy cosmic rays coming in from outside?

"We're still putting together a lot of pieces about how the local interstellar medium really interacts with the heliosphere," says Dr Ralph McNutt, a physicist at Johns Hopkins University Applied Physics Laboratory (JHUAPL) in Maryland, in the US.

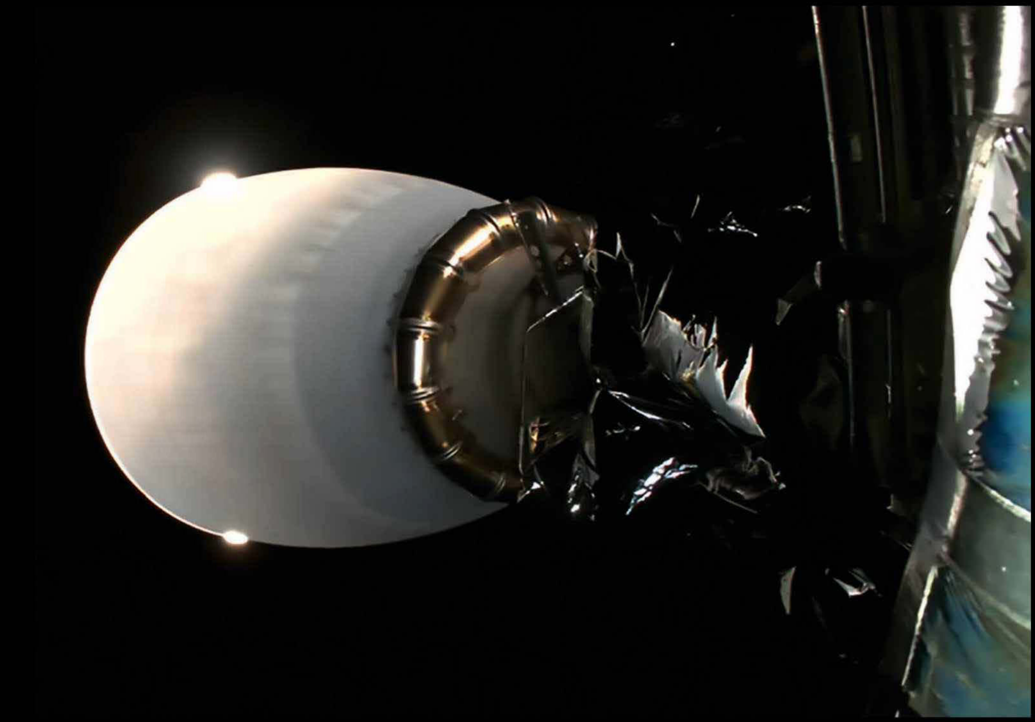
It's hoped a new $782 million (£580 million) NASA mission will help put more of those pieces together. The Interstellar Mapping and Acceleration Probe (IMAP) spacecraft was launched on 24 September 2025. And instead of spending decades travelling to the edge of the heliosphere, over the next few months it'll travel to a position about 1.5 million kilometres (one million miles) from Earth called Lagrange Point 1 (L1). Once there, unhindered by interference from Earth, IMAP can carry out its purpose: study the heliosphere, from afar.

MISSION OBJECTIVE

IMAP is a hexagonal, solar-powered spacecraft that's 2m (6.5ft) wide and looks a bit like a lollipop. It's equipped with 10 instruments designed to study the heliosphere, the Sun and incoming particles from interstellar space. It spins about four times a minute, letting its detectors scan the entire sky. Over the course of two years, it'll conduct its primary missions, continuously sending data back to Earth for scientists to pore over.

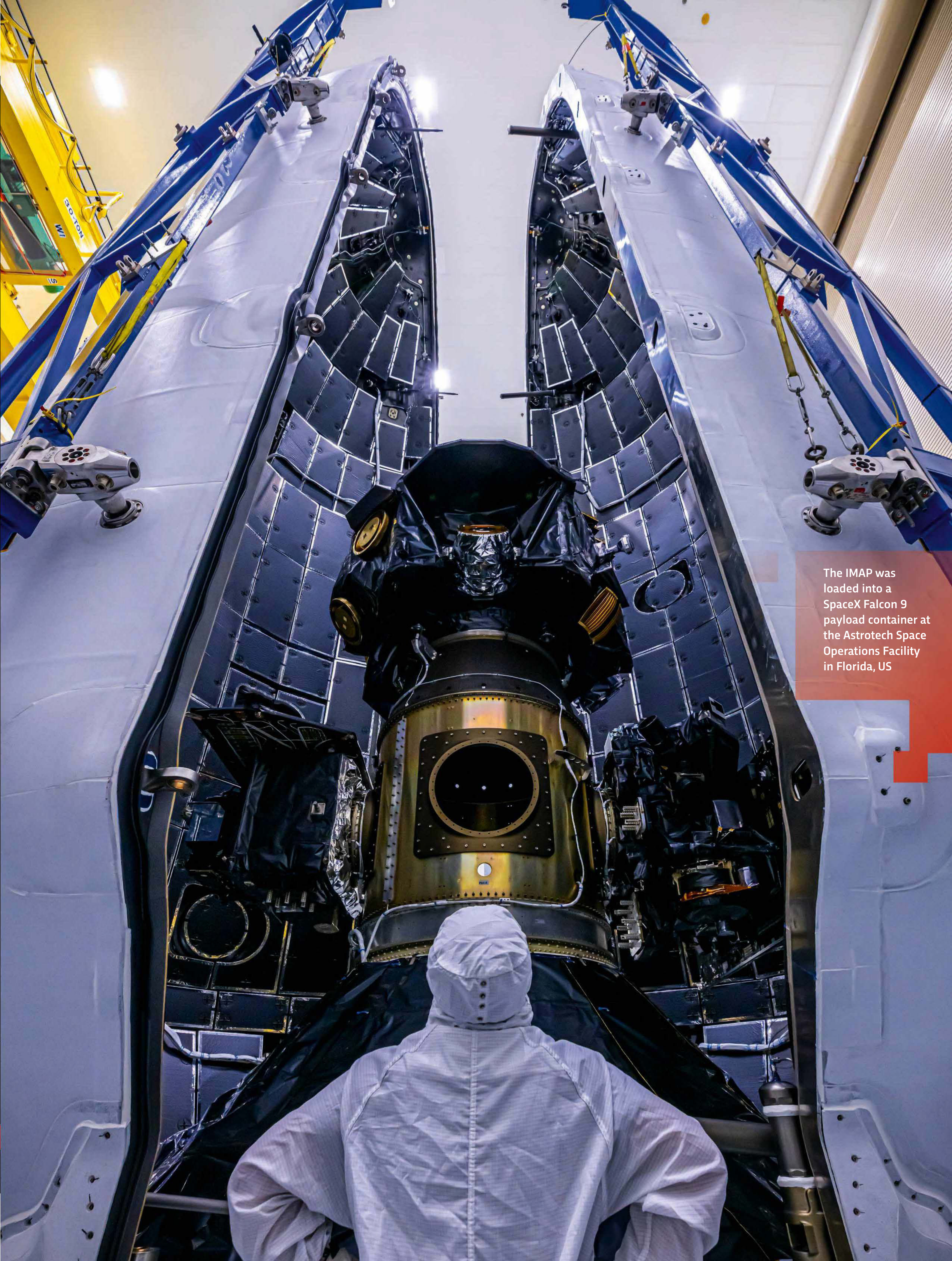
"February 2026 is the official start of the science phase of the mission," says Prof Dave McComas, mission lead at Princeton University in New Jersey, US. "The mission is nominally two years, although we have propulsion and gas to last for at least five years and probably many decades."

The purpose of the IMAP mission is to map the heliosphere around the Sun. We know from Voyager 1 and 2 that the boundary of the heliosphere and interstellar space, called the heliopause, is about 120 astronomical units (AU)

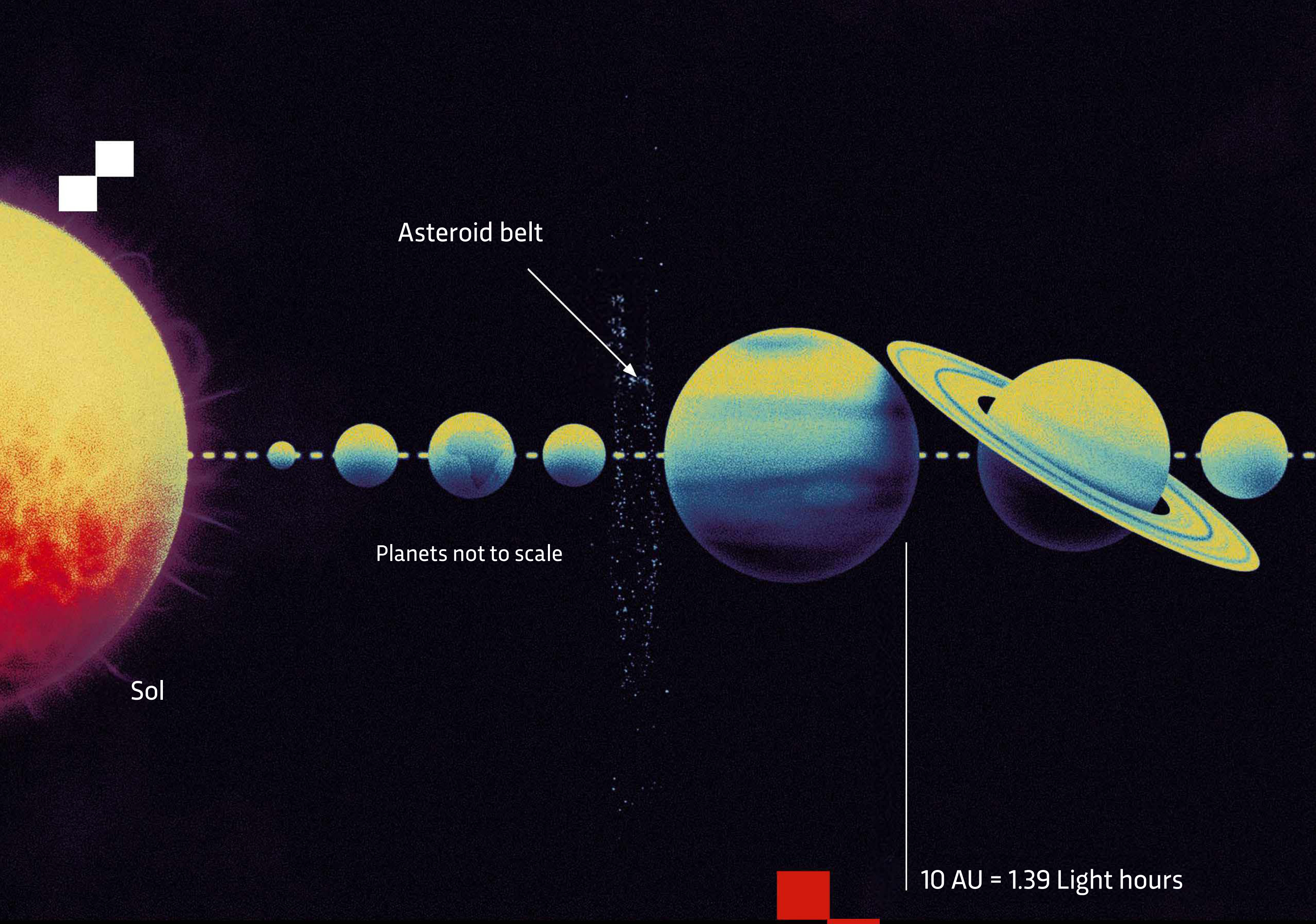


BELOW The IMAP deploying in orbit

NASA/JOHNS HOPKINS APL/PRINCETON/ED WHITMAN, BAE SYSTEMS/BENJAMIN FRY, NASA/KSC



The IMAP was loaded into a SpaceX Falcon 9 payload container at the Astrotech Space Operations Facility in Florida, US



away (120 times the Earth-Sun distance), or about 18 billion kilometres (11 billion miles). But we don't know if this is the same distance in all directions.

If it isn't, the heliosphere may not actually be a sphere. It might be more of an egg shape with a long tail. Some scientists even think it could be a croissant shape, with the Sun in the middle and two tails on either side. "We believe it'll be possible to resolve [the heliosphere's shape] with IMAP," says Dr Matina Gkioulidou, the project scientist for IMAP at JHUAPL.

To do that, instruments onboard IMAP will look at neutral atoms, tiny particles with no electric charge, coming from the heliopause. These are created when outgoing charged particles from the Sun's solar wind – a steady outflow of plasma from the Sun's outer atmosphere – crash into interstellar gas.

Because neutral atoms have no charge, they travel in straight lines. Hotter neutral atoms travel towards us faster and come from more energetic regions of interstellar space, while colder atoms originate from calmer areas. By measuring their energies and angle of arrival, scientists can model how long the atoms took to reach us. With this information, they can map the heliopause in different directions and reveal its true shape to a distance of about 300–400 AU, says McComas.

"We should be able to get the shape" in most directions, he says. In regions where the heliopause stretches beyond 400 AU, however, such as at the end furthest from the Sun, "we just won't know what that looks like," says McComas, but we should get at least an idea of the rough shape in that direction.

LIFE ON THE OUTSIDE

The nature of the heliosphere is crucial to our understanding of how we came to exist on Earth. "I'm trying to understand the properties of today's heliosphere to say if the shape has changed," says Prof Merav Opher, an astronomer and heliosphere expert at Boston University. Her research suggests Earth might have found itself beyond the heliopause in the past, during periods when the Sun was less active.

"If we were, at times, outside of the heliosphere, how did life evolve on Earth?" she asks. "It's really critical for us to understand which type of radiation Earth would have encountered."

A prior mission has already explored the shape of the heliosphere. NASA's Interstellar Boundary Explorer (IBEX) launched in 2008 and is still operational today. It gave us our first hint that the heliosphere wasn't a perfect sphere and looked slightly lopsided, but its instruments are low resolution, giving us only a blurry image of the heliosphere. IMAP should give us a much clearer picture.

"IMAP is a big step up over IBEX," says McComas. "[IBEX is] a very small mission. But you need a big, strong mission with the resolution and sensitivity to put together the full picture."

> "IF WE WERE, AT TIMES, OUTSIDE OF THE HELIOSPHERE, HOW DID LIFE EVOLVE ON EARTH?"

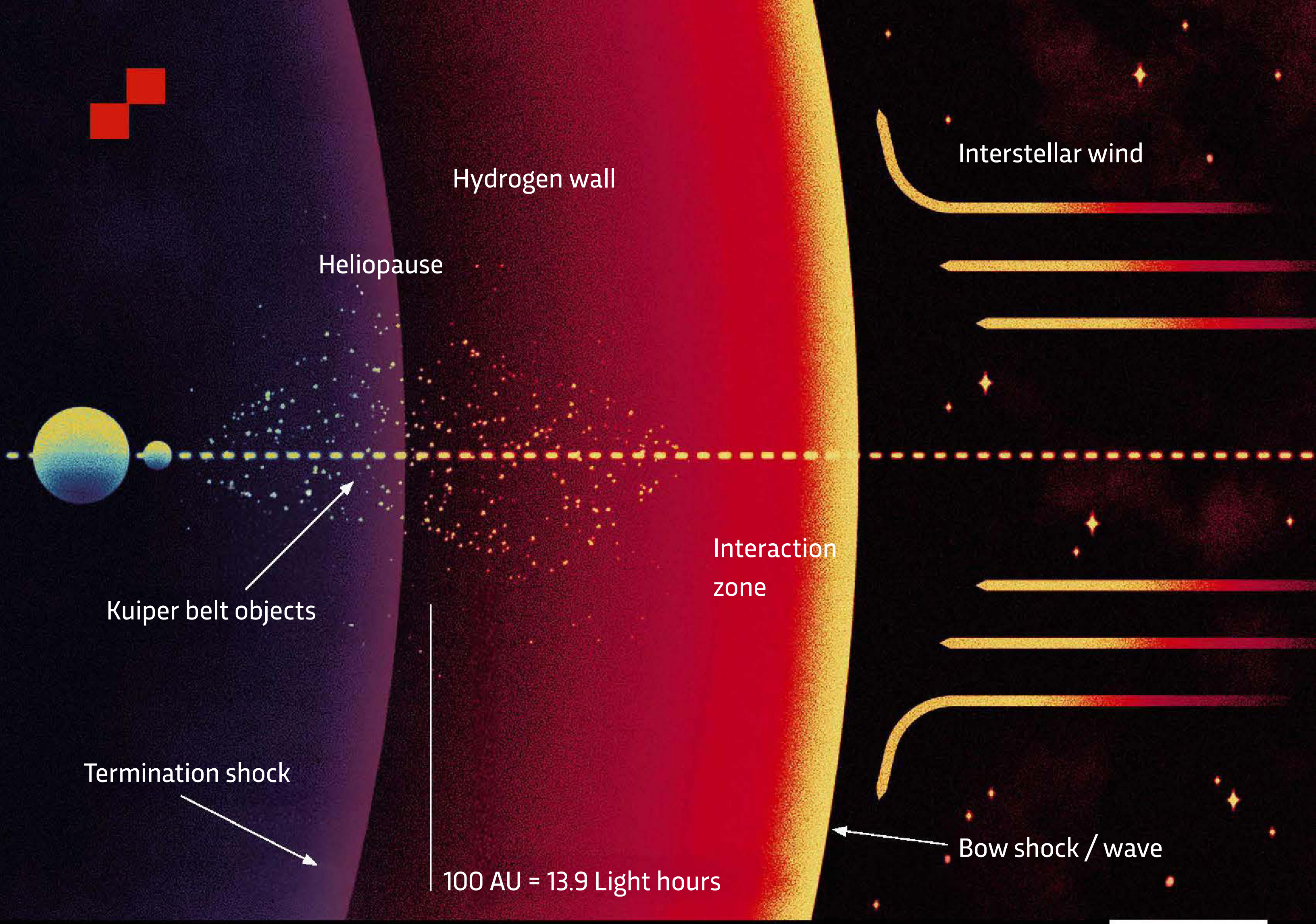


ILLUSTRATION: MAGIC TORCH

IBEX also revealed something unusual: there seems to be a band of neutral atoms surrounding the heliosphere, called the IBEX ribbon, possibly caused by the interstellar magnetic field. “Some of these energetic neutral [atoms] are in the shape of a ring in the sky,” says McNutt. IMAP should give us a better understanding of this ribbon and exactly how it’s produced.

DUST IN THE WIND

It’s not just the heliopause that IMAP will study, however. One of its other instruments, the Interstellar Dust Experiment (IDEX), will be trying to collect dust grains arriving from interstellar space. We know that there are tiny grains of dust drifting between the stars because we’ve seen their infrared glow – some spacecraft have even captured interstellar dust before – but we only have a small number of detections.

It’s hoped IMAP will collect hundreds of incoming grains of dust, both from interstellar space and from the interplanetary space between the planets of the Solar System. That’s possible thanks to the size of IDEX, which has a collection area comparable to an A4 sheet of paper. “We have this huge dust collector,” says McComas. “We should get about 100 [interstellar] dust grains in the first year alone. That would be more dust grains than all of humanity has collected in the space age.”

IDEX will measure the size, velocity and energy of the incoming dust, telling us more about its formation and journey to us. Each grain will be broken up inside the instrument, striking an ultra-pure gold surface where they become vaporised, breaking down into atoms with either a positive or negative electrical charge. A detector will then identify the elements present in the dust fragments, helping scientists deduce where they likely came from – exploding stars, for example, or other cosmic explosions.

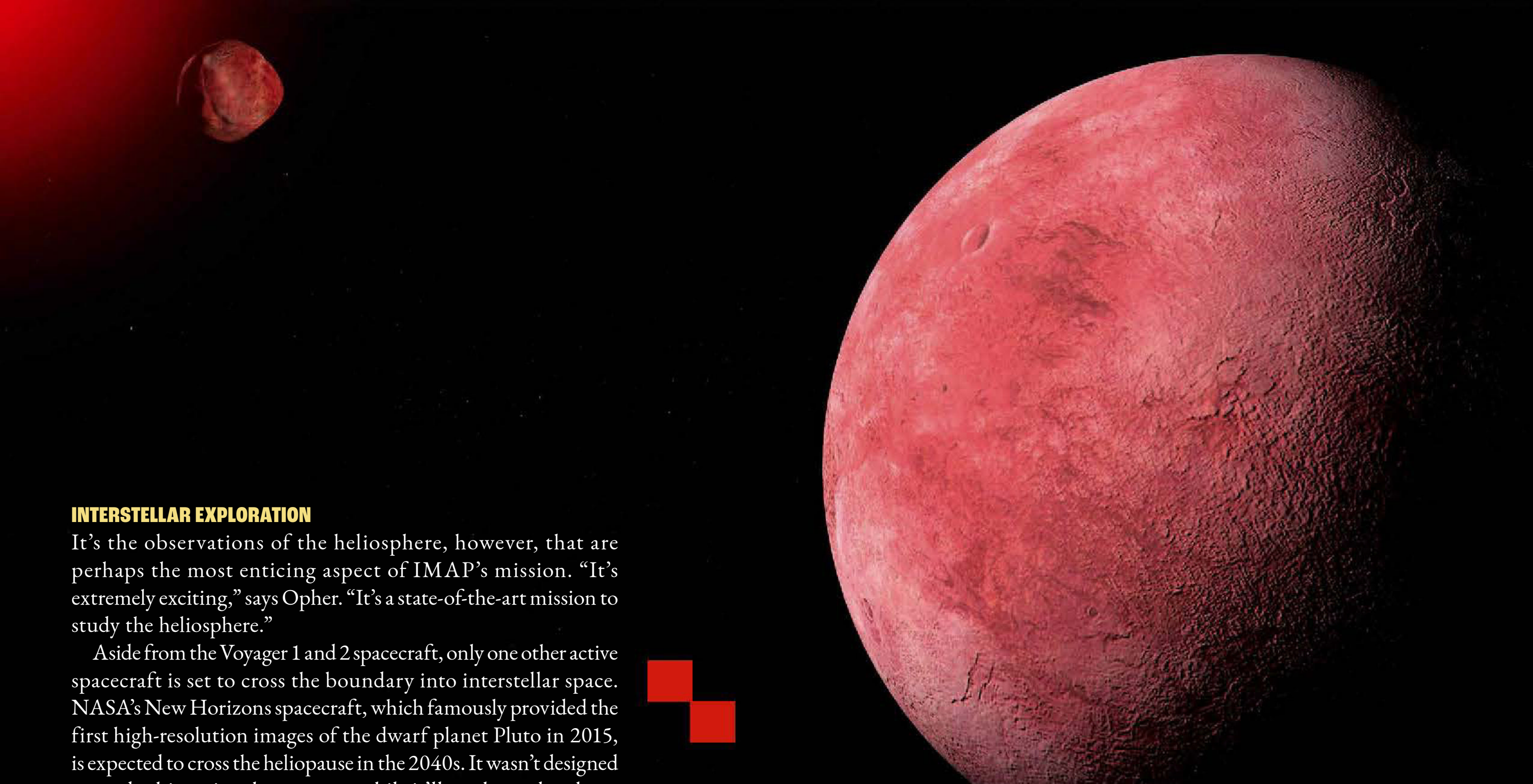
The last key goal of IMAP is to measure the solar wind at L1, including how it interacts with Earth’s magnetic field, and give us a better understanding of so-called space weather. Several of its instruments are designed to track energetic particles from the Sun that could damage satellites or cause geomagnetic storms and threaten our way of life here on Earth.

“The solar wind flies everywhere in space,” says Gkioulidou. “IMAP will measure all these particles as they’re coming out of the Sun.”

Those measurements could be useful not just for tracking space weather on Earth, but for future human missions to the Moon and Mars (both China and the US have plans to land humans on the lunar surface by 2030). Astronauts travelling that far will spend prolonged amounts of time outside Earth’s protective magnetic field and will be subjected to more solar radiation.

“If there’s an event that could be dangerous to astronauts on the surface of the Moon, [IMAP’s data] will feed into that forecast,” says Jamie Favors, director of NASA’s Space Weather Program in Washington, DC.

For humans going further afield, “on that journey to Mars, there may be months in deep space [with] no protection,” he says. “There are things about the [space] environment we don’t know.”



ABOVE An illustration of the dwarf planet Quaoar with its orbiting moon, Weywot. Both are located in the Kuiper belt, a doughnut-shaped region of icy objects and dwarf planets that orbit the Sun beyond Neptune

It's the observations of the heliosphere, however, that are perhaps the most enticing aspect of IMAP's mission. "It's extremely exciting," says Opher. "It's a state-of-the-art mission to study the heliosphere."

Aside from the Voyager 1 and 2 spacecraft, only one other active spacecraft is set to cross the boundary into interstellar space. NASA's New Horizons spacecraft, which famously provided the first high-resolution images of the dwarf planet Pluto in 2015, is expected to cross the heliopause in the 2040s. It wasn't designed to study this region, however, so while it'll teach us a lot about it, scientists are hopeful of one day sending new, dedicated missions into interstellar space.

One such proposal led by McNutt is called Interstellar Probe. It's an idea to send a long-lived spacecraft into interstellar space, designed to operate for generations and travel an incredible 1,000 astronomical units in about 50 years. That's roughly ten times farther than the Voyager probes have currently reached. The mission would harness a gravity assist from Jupiter to fling itself out of the Solar System, carrying a suite of advanced instruments to study interstellar dust, neutral atoms, magnetic fields and more beyond the heliopause.

"I've been convinced this is a good idea for a long time," says McNutt. "It's just a question of getting other people convinced."

In 2024, the National Academies of Sciences, Engineering and Medicine in the US – a public body that advises the US government on science, engineering and health – announced its proposal for the next decade of NASA's research into the Sun and its surrounding environment, known as the Heliophysics Decadal Survey.

The survey didn't directly recommend building Interstellar Probe, but it did say it was a compelling future mission that should be considered in the next decadal survey in the 2030s. Having such a mission to collect in-situ data (data directly from the source) would be invaluable, says Opher. "We still want a new Voyager," she says. "There's nothing like in-situ data."

It's not just NASA considering a new mission to interstellar space. China is investigating a possible mission called Interstellar Express, or Shensuo. It would send two or more probes beyond the heliopause, carrying out flybys of Neptune and other interesting objects (like the dwarf planet Quaoar in the outer Solar System) along the way. The status of the mission currently remains uncertain, but could be hugely exciting if it goes ahead.

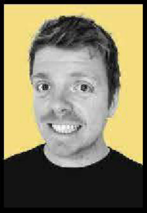
There are more speculative ideas, too. Opher is part of a team that has proposed using a fusion-based propulsion system to explore the heliosphere in a way that's simply not possible with conventional chemical spacecraft engines. "We could launch six different spacecraft in all directions of the heliosphere based on fusion propulsion," says Opher. "Something like that can bring us fast to the edges. We're trying to get to the edge in five years and then slow down to get all the data."

Whether humans, rather than just our robotic explorers, will ever venture into interstellar space seems like a far more daunting prospect. Such a mission would almost certainly carry a one-way ticket. Perhaps a bold attempt could be made to reach one of our closest stars, such as Proxima Centauri, about four light-years (approx 40 trillion kilometres) away, where astronomers are hopeful there might be interesting planets to visit.

For the time being, however, it's IMAP that's at the forefront of everyone's minds, as it sets out to study the heliosphere from a vantage point, not in the empty expanse of interstellar space, but from a quiet corner of the Solar System.

Will it reveal that the heliosphere is shaped like an egg, a croissant, or something else entirely? In the coming years, we should finally have a solid answer to this question, and plenty of others, as well as a better understanding of the boundary between our Solar System and interstellar space. **SF**

> **"SCIENTISTS ARE HOPEFUL OF ONE DAY SENDING NEW, DEDICATED MISSIONS TO INTERSTELLAR SPACE"**



by **JONATHAN O'CALLAGHAN**

Jonathan is an award-winning freelance science journalist based in the UK.

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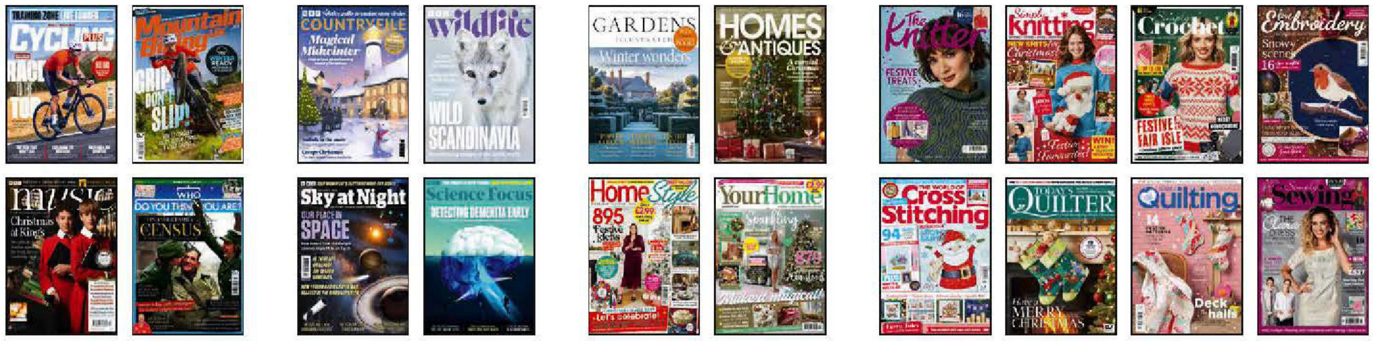

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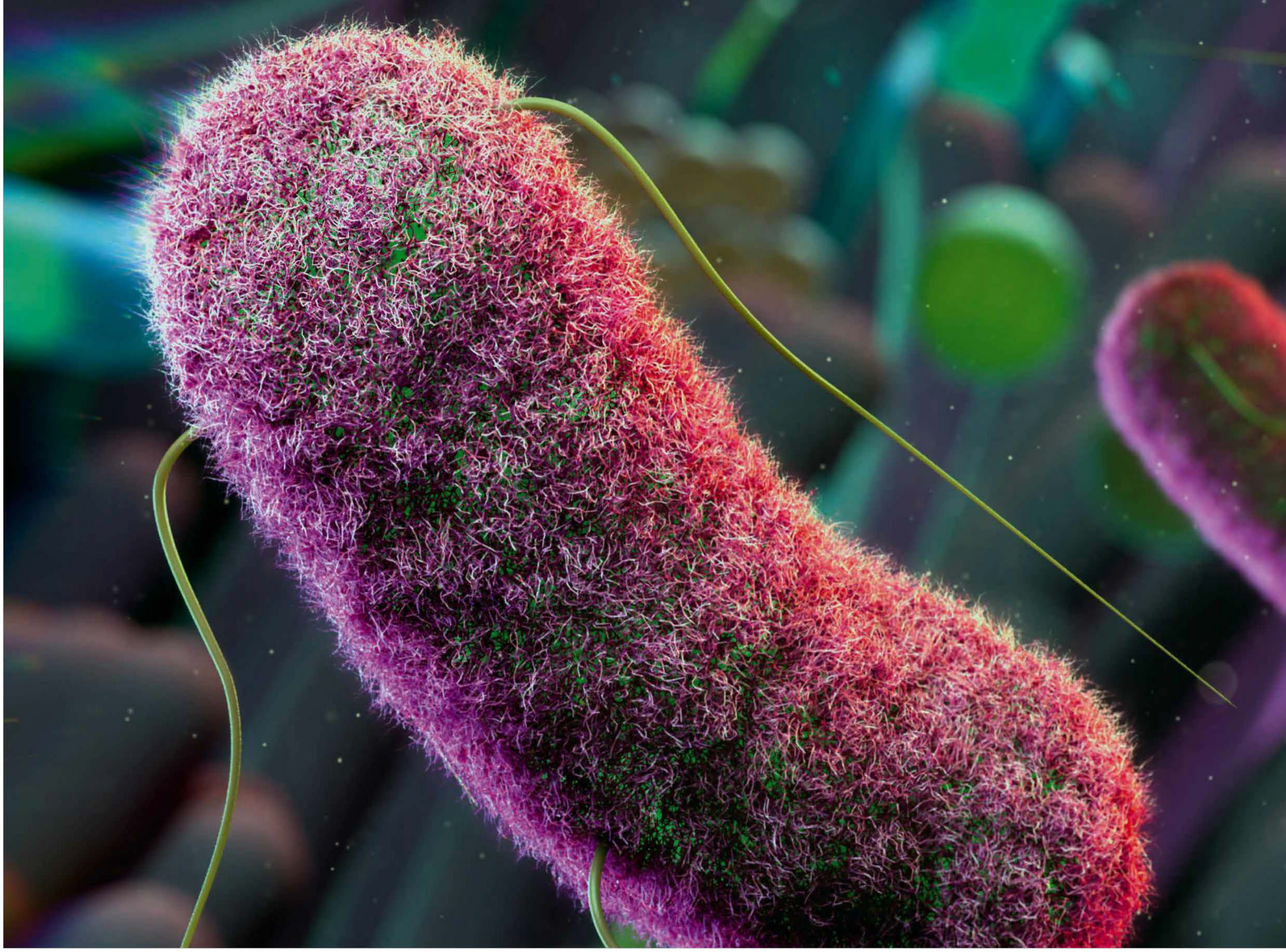
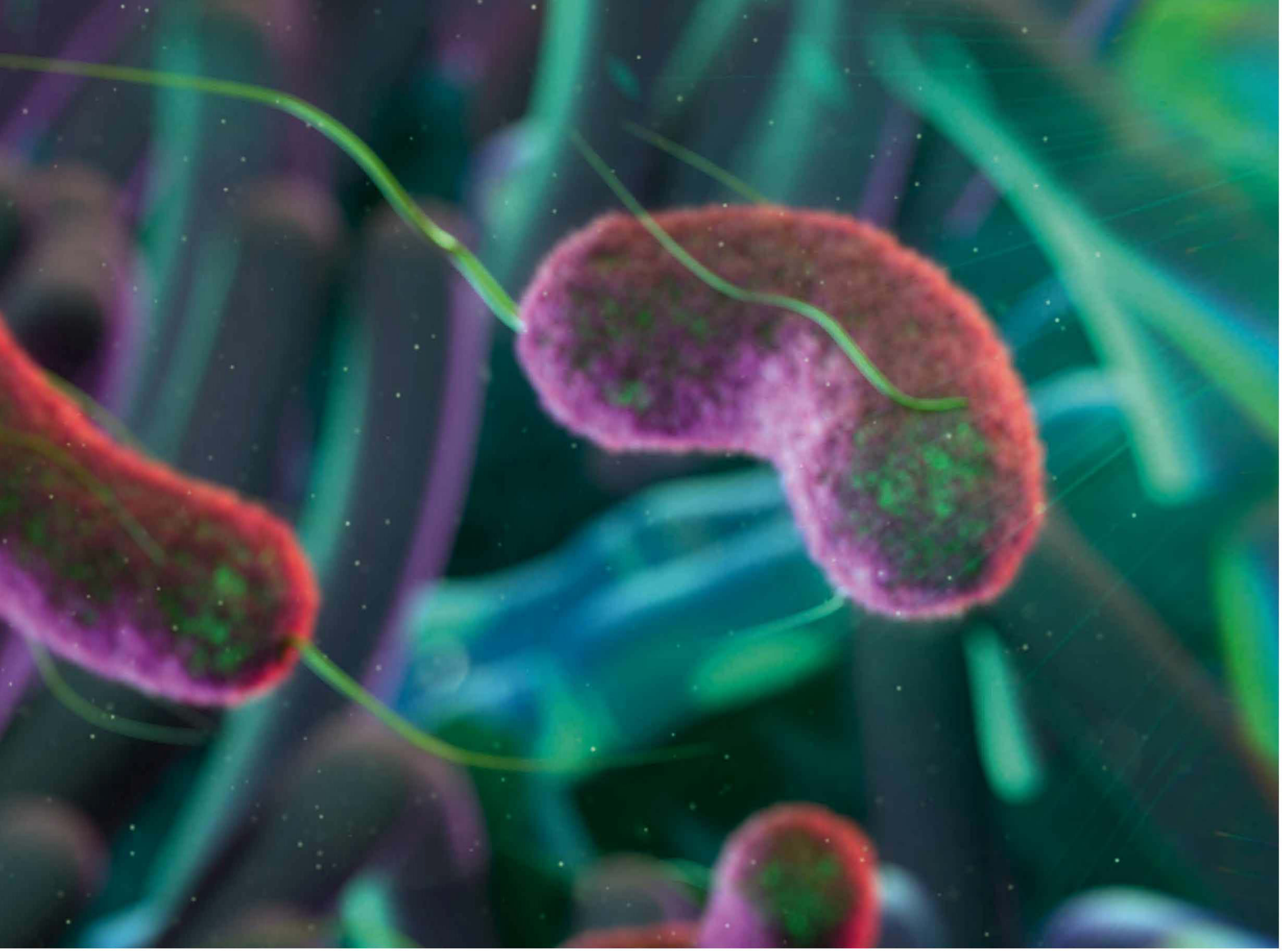


WANTED: GUT BACTERIA DEAD OR ALIVE

There are millions of bacteria living in our guts. There are millions of dead bacteria there too. And scientists are learning just how much potential the dead ones have to improve our health

by **PROF TIM SPECTOR**



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The term 'gut health' pops up everywhere these days. But while phrases such as low-carb diets, low-fat diets and high-protein snacks have come and gone, gut health is more than just a fad. Unlike previous trends, the gut-health movement is backed by solid scientific evidence gained over two decades of discovery.

We've known that there are helpful and harmful bacteria living in our guts for a long time, but it's only been during the last 20 years that we've come to realise just how important they can be. During that time, we've gained insights into the effects they have on our health – both physical and mental – and the influence that the foods and drinks we consume have on them.

This voyage of discovery is far from over, though, because up until now, our research has been concentrated on the bacteria that are living in our guts. But our gut microbiomes contain plenty of dead bacteria as well. And we're only just beginning to learn about the role that dead bacteria – postbiotics – has to play.

YOUR MICROBIOME AND YOUR HEALTH

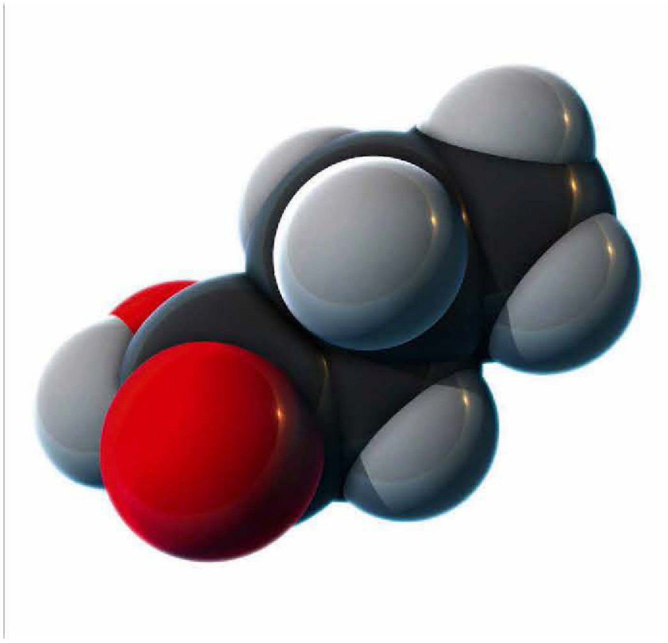
Collectively termed the microbiome, each of us has trillions of microbes that live on and in us. We are, in short, teeming with life. Although distributed widely, most microorganisms are in our gut, specifically our large intestine – the final section of our gastrointestinal tracts, right before the exit.

Until 20 years ago, scientists believed that our gut microbiome was fairly incidental and not particularly significant for our general health. We knew that the microorganisms living in our guts helped break down some of our food, but beyond that, they were considered relatively unimportant and uninteresting.

We now know, however, that the microbes in our guts – the bacteria, fungi, viruses and more – are far more important for our health than we could have previously imagined. It's becoming ever clearer that good gut health is good overall health.

Our microbiome evolved within us as we evolved within our environment. Since the dawn of life, this mixture of microbes and multicellular life has developed in tandem with us. It's impossible to separate one from the other – you and your microbiome are a superorganism that's been slowly developing together to be mutually beneficial.

As technology has advanced, we've been able to dig deeper into the gut microbiome, exploring the role of individual species within it. It's now clear that harbouring a diverse range of 'good' gut bacteria is associated with better overall health. Conversely, if your gut microbiome has low diversity of species and higher numbers of 'bad' bacteria,



LEFT A molecular model of butyric acid, a short-chain fatty acid found in milk and butter

you're at an increased risk of experiencing poorer health.

Recently, we've found associations between a disrupted gut microbiome and a wide range of health conditions, including type 2 diabetes, obesity and even neurodegenerative conditions, like Parkinson's and Alzheimer's disease. Whether a disrupted gut microbiome causes these health conditions or is affected by them, or both, remains an open question, but the ties between gut health and disease are now inescapable.

PRE- AND PROBIOTICS

As the science of the gut microbiome develops, and news of its near-miraculous importance has reached the mainstream, many of us are keen to find the most effective ways of supporting and improving our society of internal microorganisms. In this regard, the most important factor is fibre, a macronutrient that 90 per cent of people in the West are deficient in.

Previously known as 'roughage', doctors believed it helped 'keep you regular'. This is certainly true, but fibre's importance goes well beyond the frequency of your bowel movements. Fibre, present in all plants, is impervious to your digestive enzymes. This means that it makes the journey through your intestines intact, eventually reaching your colon, where the vast majority of your gut bacteria live.

Once there, fibre is fermented by gut microbes, keeping them well fed and happy. In return, gut bacteria produce a suite of bioactive compounds that support health. Each bacterium is akin to a miniature pharmacy that dispenses medicine in return for plant fibre.

One of the most well-understood microbial products of fibre fermentation are short-chain fatty acids (SCFAs), including propionate, butyrate and acetate. SCFAs provide energy to the cells that line your gut, helping to ensure the lining stays healthy and doesn't leak. SCFAs also play a part in blood glucose control, reducing inflammation and more. Aside from producing SCFAs, as bacteria ferment fibre, they also produce gases and other compounds, such as indole-3-acetic acid, as well as thousands of other chemicals, including vitamins and neurotransmitters.

So, fibre is essential for gut health, and simply by increasing your intake you can quickly improve the health of your gut microbiome. But fibre isn't the only way to support gut health. There are also prebiotics and probiotics.

Prebiotics, which include fibre, can be considered the soil of your gut garden, nourishing your gut bacteria. Meanwhile, probiotics are microbes that help bolster your internal community of microbes. Products that contain both pre- and probiotics are referred to as synbiotics. Fermented plant products, such as kimchi and sauerkraut, are good examples of synbiotics, because they contain fibre from the plants plus live microbes.

Although probiotic supplements are now mainstream and increasing in popularity, evidence supporting their health benefits is still lacking in most cases, especially in the prevention of disease. This is partly because they tend to contain just one or a small handful of bacterial species.

In general, the species are selected not because of their efficacy, but because it's legal to use them and they're easy to culture. Fermented foods, on the other hand, contain much more bacterial variety and, therefore, are much more likely to support the diversity and health of your gut's internal ecosystem.

DEAD BACTERIA

More recently, a new -biotic has entered the scene: postbiotics. In short, postbiotics are dead microbes, parts of dead microbes, products produced by microbes or a combination of all three.

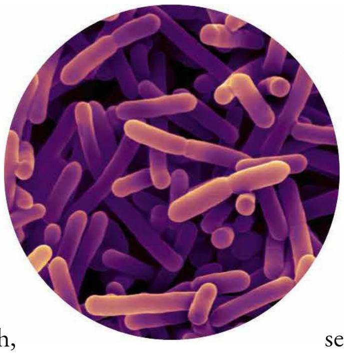
Recently, their benefits have been investigated and I've been surprised and excited by the results. It seems that a dead microbe still has the ability to support overall health.

How can a dead microbe impart health benefits? It's a fair question and, I admit, I wasn't initially convinced. It helps to imagine them like vaccines that contain inactive microbes. Although the pathogen in the vaccine is dead, the microbe still has a cell wall with proteins and triggers a response from your immune

> "LATER RESEARCH FOUND THAT DEAD BACTERIA FROM VARIOUS SPECIES WERE EFFECTIVE AGAINST A RANGE OF MALADIES IN CHILDREN"

system, priming it for the next time you encounter the pathogen. In a similar way, postbiotics, despite being dead, can support your gut microbiome.

Just a few years ago, researchers would use inactivated microbes as a control in experiments. In effect, they were comparing the effectiveness of probiotics to postbiotics. After some studies unearthed surprising results from the postbiotic placebo arm of the research, scientists in Japan dug a little deeper, concluding that a heat-inactivated *Lactobacillus pentosus* strain could increase participants' immune protection against the common cold.



Later research found that dead bacteria from various species were effective against a range of maladies in children, including diarrhoea and sore throats, while a large study conducted in Germany showed that dead *Bifidobacterium bifidum* could relieve symptoms of irritable bowel syndrome (IBS). These beyond-the-grave health benefits seem almost unbelievable at first glance, but there's now enough evidence that we can no longer ignore the ghosts of long-dead bacteria.

The study of postbiotics is still in its infancy, but I find the idea that dead or inactivated microbes can support health to be quite a relief. I always wondered whether the probiotics in sourdough might still benefit me after being baked alive, or whether sauerkraut's microbes could still support health after being boiled in a stew. It seems that they can.

One small study, for instance, compared heat-treated sauerkraut with standard 'live' sauerkraut, concluding that both forms eased IBS symptoms to a similar degree.

To date, the most studied postbiotic is *Akkermansia*, a microbe that helps keep the barrier between our gut and bloodstream in good condition, thereby preventing toxins and other nasties from leaching into our blood. *Akkermansia* is associated with better metabolic health, blood glucose control and weight loss.

One of my colleagues, Patrice Cani, professor of physiology, metabolism and nutrition at the University of

ABOVE Kimchi, a synbiotic, contains both pre- and probiotics

LEFT *Bifidobacterium bifidum* seen under a scanning electron micrograph

SCIENCE PHOTO LIBRARY X2, GETTY IMAGES

ABOVE Unhealthy guts (left) typically have a less varied population of bacteria

ABOVE LEFT Different fibre from a wide range of fruits and vegetables supports a healthier, more resilient gut

> **"IT'S BECOMING EVER CLEARER THAT DEAD MICROBES CAN IMPART SIGNIFICANT BENEFITS"**

Louvain in Belgium and visiting professor at Imperial College London, was surprised to find that dead *Akkermansia* had significant anti-obesity and anti-diabetic properties in both mice and humans. Even more surprising was that the dead version was more effective than the live microbe.

THE FUTURE OF FOOD

Precisely how postbiotics can exert health benefits from beyond the grave is a topic of much scientific interest. There are likely a number of mechanisms at work, many of which may be working in unison.

One such mechanism relies on microbes' community spirit. Fermented foods contain a wide range of species, but they don't exist in a vacuum. They're intimately linked, sharing food, competing for resources and protecting themselves from threats. This tight-knit community, like those found in fermented dairy, often produces rigid sugar structures known as exopolysaccharides, which serve to protect the entire ecosystem, despite being formed of highly disparate species. We could learn a lot from these cooperative lifeforms.

Research now suggests that these protective structures, rather than the microbes themselves, may help reduce inflammation and protect your gut barrier. So, even after death, the protective structures these microbes build can benefit human health. Some animal research has also shown that dried and powdered fermented foods, which contain exopolysaccharides, can reduce blood pressure and lower cholesterol. There's even evidence that they may help protect against cancer.

Although this is a rapidly developing field, it's becoming ever clearer that dead microbes can impart significant benefits, although precisely how will take a little more unpicking. This is clearly good news for people who cook with fermented foods, but it also opens the doors to innovation in food science.

As it stands, only a handful of microbes are allowed to be used in food products. Adding live bacteria to food is, rightfully, highly regulated. From a safety point of view, it's important that manufacturers don't add mysterious, unstudied species to food products and probiotics. That's asking for trouble. It's a similar situation for researchers: using live microbes in studies is heavily regulated for good reason. These restrictions become much less burdensome once a bacterium is deceased and can't replicate, however.

The recent discoveries that a dead microbe may be just as useful as a live one opens the door for innovative food products and more research. Although I was sceptical initially, I now believe that postbiotics will be a mainstay in the future of functional food. Evidence is mounting that both live and dead microorganisms can support our gut and overall health, and I'm excited to see what we learn next about the wonders of microbes. **SF**

by **PROF TIM SPECTOR**

Tim is a Professor of Genetic Epidemiology at King's College London, co-founder of ZOE Ltd and an author. His latest book Ferment: The life-changing power of microbes *is out now (Jonathan Cape, 2025).*

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CRACKING CHOLESTEROL

What really causes your blood fats to skyrocket? Groundbreaking discoveries have delivered surprising new answers and exposed a deeper biological mystery

by **SANDRINE CEURSTEMONT**

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When you think of cholesterol, what's the first food that comes to mind? For many, the answer is eggs. And for good reason: their yolks contain relatively high amounts of the waxy fat found in our blood and decades-old studies warned that eating too many could send your cholesterol levels soaring.

As any doctor will tell you, that would be a very bad thing. High cholesterol raises your risk of cardiovascular disease – the world's leading cause of death – as well as stroke and dementia.

According to several recent papers, however, eggs may not deserve their bad reputation. In fact, one Australian study suggests that eating two a day could *lower* your levels of 'bad' low-density lipoprotein (LDL) cholesterol – the kind that clogs your arteries.

"For your average person, eating a few eggs a day won't increase their cholesterol," says Dr Nick Norwitz, a researcher at Harvard Medical School in Boston.

How could this be? Well, as emerging studies have demonstrated, dietary cholesterol – the cholesterol that's in our food – has little bearing on the LDL cholesterol in our bodies.

Instead, researchers have uncovered what really matters in your diet, and which foods actually make the biggest difference.

FAT TRUTHS

Somewhat confusingly, LDL cholesterol isn't actually all bad – your body needs some in order to function properly. It's a key component of the outer membrane of every cell, for example, and is needed to make vitamin D and certain hormones such as oestrogen and testosterone.

You don't need to get this helpful cholesterol from your diet, however. The liver makes all the cholesterol your body requires. And if you eat cholesterol-rich foods, it automatically scales back its production.

"If you increase your dietary cholesterol intake, that suppresses your liver's cholesterol production," says Prof Jon Buckley, a researcher at the University of South Australia in Adelaide, Australia. "So it balances out."

In other words, your liver can regulate dietary cholesterol without much trouble. Through a finely tuned feedback system, it senses changes in intake and compensates to maintain stable levels in the bloodstream.

The real problem, as Buckley's research shows, isn't dietary cholesterol, but saturated fat. The distinction between the two substances isn't always obvious, since many foods that are high in cholesterol are also rich in saturated fat – red meat, butter, cheese and fried foods, for example.

"DIETARY CHOLESTEROL HAS LITTLE BEARING ON THE LDL CHOLESTEROL IN OUR BODIES"

Eggs and shellfish, however, are the exception, being high in cholesterol and low in saturated fat.

To untangle the effects of each, Buckley's team ran a five-week study with 61 healthy volunteers, comparing diets with different levels of cholesterol and saturated fat.

The results: the more saturated fat participants ate, the higher their blood cholesterol climbed. But the amount of cholesterol in their food didn't seem to matter.

"This was the first study that had really been designed in a way that we could tease out those effects so convincingly," says Buckley.

HEART-SMART MENU

As Buckley's results suggest, reducing the amount of saturated fat in your diet can help limit bad cholesterol. If you're tempted by a cooked breakfast, for example, he suggests swapping out some of the bacon and sausages, which are high in saturated fat, for extra eggs to help keep blood cholesterol levels low. Cutting them out completely would be a positive step too, but may be more difficult.

"The best thing is to try and make very small changes that are achievable and sustainable, rather than trying to change a whole diet," says Buckley.

Switching up the type of fat in your diet can also help manage bad cholesterol. While the body requires some fat to function

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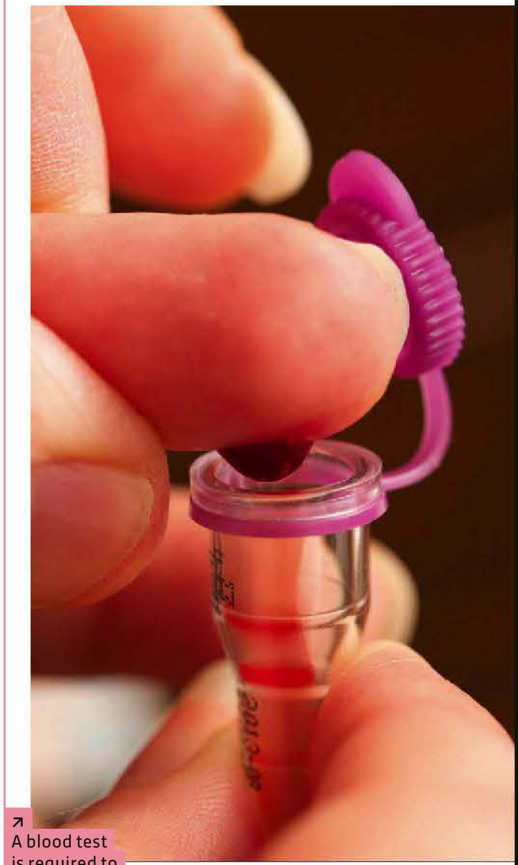

↗ A blood test is required to check your cholesterol levels

MEASURE YOUR CHOLESTEROL

The most accurate way to check your cholesterol is with a blood test. Some clinics and pharmacies offer quick finger-stick tests that give results in minutes, but most require a blood sample from your arm after fasting for 9–12 hours.

Your results will show levels of 'bad' low-density lipoprotein (LDL) cholesterol, which can build up in arteries, and 'good' high-density lipoprotein (HDL) cholesterol, which helps remove it. A total cholesterol reading combines both, plus some of your triglycerides – another type of blood fat linked to heart disease.

According to Johns Hopkins University, in the US, healthy LDL levels in adults are below 100 milligrams per deciliter (mg/dL). Levels between 160–189mg/dL are high, and over 190mg/dL may require treatment.

Levels below 40mg/dL are classed as very low. It's unclear if this is harmful, although some studies suggest possible links to depression and diabetes.

↗ *Bifidobacteria*, a probiotic bacteria in your gut, may play a role in reducing cholesterol

– including for the absorption of vitamins A and D – most people consume excessive saturated fat and too little polyunsaturated fat, which is abundant in walnuts, oily fish such as salmon and plant-based oils.

"Polyunsaturated fat actually reduces LDL cholesterol," says Javier Gonzalez, a professor at the Centre for Nutrition, Exercise and Metabolism at the University of Bath, in the UK. It does this by encouraging the liver to remove LDL particles from the bloodstream more effectively, lowering your overall levels.

Conversely, Gonzalez adds, saturated fat is twice as potent as polyunsaturated fat, meaning that it can raise your cholesterol levels to a greater extent than polyunsaturated fat can reduce them.

Yet it's not just about the fats you eat – fibre plays a key role too. As recently confirmed in a study from India, eating a diet rich in fruits, oats, nuts and seeds can keep blood cholesterol levels in check.

These foods are particularly high in soluble fibre, which binds to bile acids (compounds made from cholesterol) in the gut and carries them out of the body. To replace those lost bile acids, the liver draws more cholesterol from the blood, helping to keep levels healthy.

Insoluble fibre, which is found in foods such as whole wheat, brown rice and many vegetables, has other digestive benefits, but a smaller effect on cholesterol.

Typically high in both polyunsaturated fat and fibre, a plant-based diet is an effective way to keep blood cholesterol within a healthy range. In fact, a recent large-scale trial where participants were randomly assigned different eating patterns found that those on vegetarian or vegan diets recorded lower cholesterol levels than people who had meat in their diets.

METABOLIC MYSTERY

Cut back on the bacon and eat more oats – simple enough, right? Yet cholesterol is still only partly understood, and emerging research could point to even more effective dietary approaches in the years ahead.

One area stirring debate is the ketogenic diet. Still in its early days of research, this low-carb, high-fat, moderate-protein diet is raising unexpected questions about how the body handles fat and cholesterol.

Let's be clear: doctors aren't recommending the keto diet as a shortcut to better cholesterol – quite the opposite. At present, it's mainly prescribed under medical supervision for conditions such as epilepsy and diabetes.

Even so, many people turn to keto for weight loss. The idea is that when the body runs low on carbohydrates, it switches to burning fat for fuel (a state known as ketosis), which may also reduce hunger. Evidence for its long-term benefits is mixed and side effects (including constipation, nutrient deficiencies and fatigue) are common. Still, for researchers, the diet offers a unique window into how cholesterol levels respond to a dramatic dietary change.

One such researcher is Nick Norwitz, the Harvard Medical School researcher quoted earlier. When he was following a keto diet to help manage a chronic bowel condition, he noticed that something strange happened to his blood cholesterol levels. Although he was eating healthily, consuming very little saturated fat and lots of leafy greens, fatty fish and fibre, his cholesterol levels skyrocketed to five times over what's considered healthy (see 'Measure your cholesterol', opposite, for more on healthy levels of cholesterol).

"This was scary and very bizarre because there was nothing like this in the literature," says Norwitz, who is lean and metabolically healthy.

Norwitz's personal experience drove him to start researching why he and other people were having this response. In recent work, he and his team examined how a low-carb diet affected cholesterol levels in people with different body mass indexes (BMIs) – a measure of whether someone's weight is healthy for their height – to see if this could explain the pattern.

Their analysis combined data from 41 studies reporting both BMI and cholesterol outcomes in people following low-carb diets. The results were striking: cholesterol

> **"WORKING OUT WHY SOME ROUTES TO WEIGHT LOSS SEND CHOLESTEROL SOARING COULD HELP UNCOVER HOW THE WHOLE SYSTEM REALLY WORKS"**

levels tended to rise in leaner participants, but stayed largely stable in those who were overweight. It's a counterintuitive finding, given that slimmer people are generally considered fitter and healthier.

So, could another hidden factor be driving cholesterol levels? Could, perhaps, the answer be found not in what we eat, but in how our bodies process it?

"This is one of the areas we don't fully understand," says Norwitz. "It might be linked to switching from a carb and sugar-based metabolism to a fat-based metabolism, and your body using cholesterol-enriched particles to help traffic that fat."

Norwitz was also curious to see if simply adding carbs back into his diet could lower his blood cholesterol. He decided to supplement his keto diet with cookies for 16 days. Later, after returning to the keto diet for three months, he tried a six-week course of statins to compare the effects. Surprisingly, his cholesterol dropped more during the cookie phase than it did while taking the medication.

Of course, as Norwitz acknowledges, he is a sample size of one – and the scientific consensus will remain sceptical until far more rigorous, large-scale studies are carried out. Still, his case hints that there's a lot about cholesterol that we still don't understand.

"This will not generalise to your average person, but the point is context," says Norwitz. "The fact that you could accurately predict that cookies will effectively lower cholesterol in one patient highlights just how much is unknown about cholesterol metabolism in humans."

PROFOUND RESULTS

Norwitz's case is certainly unusual – and hasn't yet been replicated in a large, rigorous study of other lean people. That's partly because most keto research focuses on the diet's effects on weight loss and cholesterol, rather than the underlying metabolic mechanisms.

And weight loss itself can strongly influence your cholesterol levels. Studies show that overweight adults who lose 5–10 per cent of their body weight through a balanced, calorie-controlled diet and exercise see clear improvements in their cholesterol levels.

↖ LDL-reducing polyunsaturated fat can be found in salmon

Gonzalez and his colleagues found, however, that the story changes on keto. In their study, one group of participants cut back only on sugar, while another restricted almost all carbohydrates – the defining feature of a keto diet. A third control group ate a balanced mix of fats, proteins and carbohydrates.

Both the low-sugar and keto groups lost a similar amount of weight, but their blood cholesterol told two completely different stories. In the low-sugar group, cholesterol levels fell; among those on keto, they surged dramatically.

It's tempting to simply focus on which diet works best here, but the results point to something more profound. As Gonzalez says, "It seems like the way in which you achieve weight loss can influence your cholesterol response."

In other words, working out why some routes to weight loss send cholesterol soaring could help uncover how the whole system really works – and how to manage it more effectively.

As Gonzalez says, part of the reason keto can send cholesterol soaring may be straightforward: the diet involves eating more saturated fat and less fibre – both known risk factors for high cholesterol. But his team also suspects there's more to the story. For instance, changes in the gut microbiome.

Trillions of microbes live in your digestive tract and are known to play a big role in metabolism, body weight and appetite. Gonzalez found that a particular type of bacteria, called *Bifidobacteria*, was reduced in people eating a keto diet compared to the control diet.

"We know from other studies that *Bifidobacteria* play a role in cholesterol metabolism," he says. "We have a project starting very soon to understand whether fibre intake can help mitigate the increase in LDL cholesterol on a ketogenic diet."

Gonzalez's study uncovered another layer of complexity. Participants following the same diet often experienced markedly different changes in their cholesterol levels. This suggests that there isn't a single diet that will help everyone maintain healthy cholesterol levels and that nutritional advice may need to be personalised.

In some cases, genetics could play a role, which has been examined in people eating a standard mixed diet. A few different genes can influence the liver's ability to clear cholesterol from the blood, for example, or how well it's absorbed from the food you're eating.

Gonzalez and his team are now planning to study this in more detail. "We will look at those genes and markers of absorption and synthesis to understand that better," he says.

Norwitz also believes there's an open question over how personal factors might influence the risk that high cholesterol actually plays in cardiovascular health. Because most cholesterol studies focus on people with obesity or diabetes, it can be difficult to tease out how cholesterol behaves in other people.

"It's not that cholesterol isn't important," he says. "But it does absolutely need to be taken into the broader context of a person's health profile and not be looked at through the keyhole."

Cholesterol clearly has more secrets to give up and it may be some time before researchers can explain every twist in its story. In the meantime, the best approach is the boring but reliable one: limit saturated fat, load up on fibre and healthy oils – and, yes, you can still enjoy your morning eggs. **SF**

by **SANDRINE CEURSTEMONT**

Sandrine is a freelance science journalist, whose work has been published by BBC Future, National Geographic *and others.*

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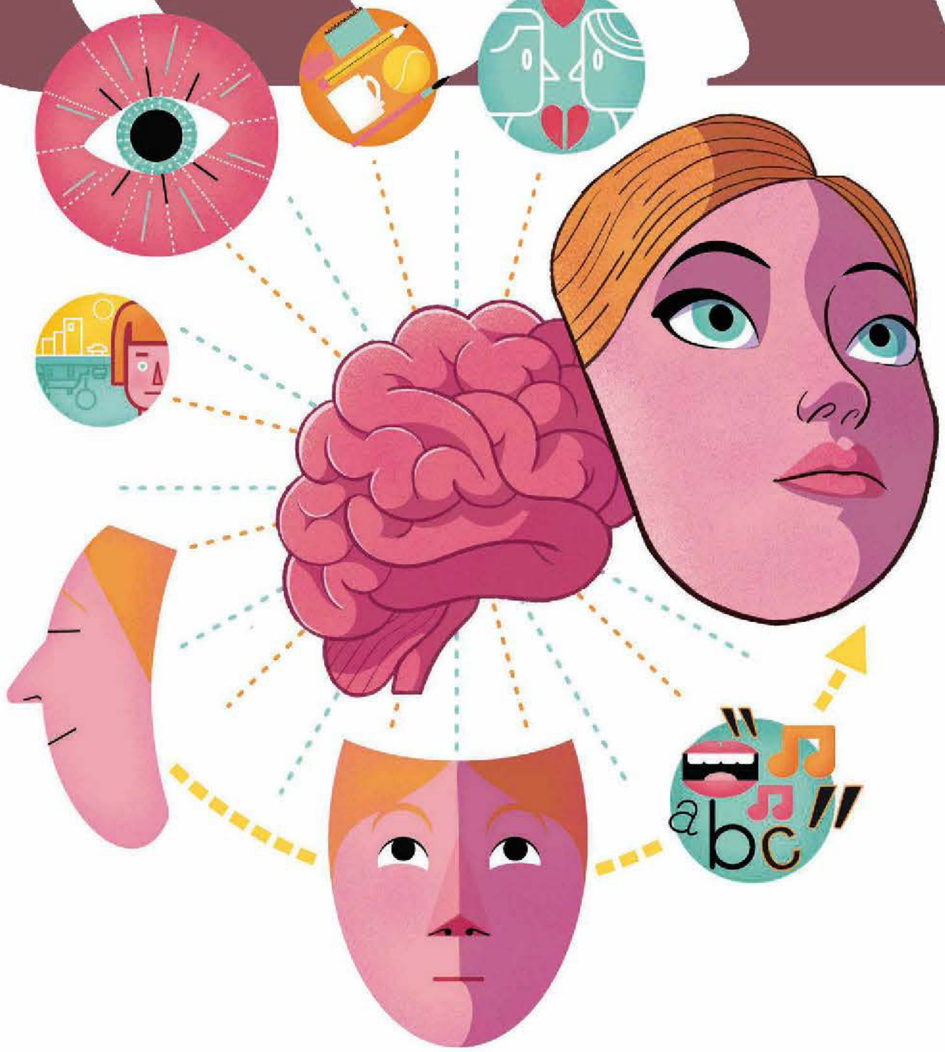


FRANK ROSS, SOUTHAMPTON

HOW DOES MY BRAIN CREATE MY SENSE OF SELF?

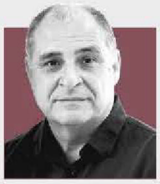
Your ability to experience a stable, coherent sense of self is truly a miracle. Each of us experience profound changes across the course of our lifetime, growing from infants into adults, learning new things, forgetting others, forging fresh relationships and losing old ones – all interspersed with prolonged interludes of dreams and blankness each and every night. And yet, throughout it all, we still have a sense of being the same person. Ultimately, this comes down to an ongoing construction process operating in the brain, which is probably more malleable and fragile than you might think.

Classic research in the latter half of the 20th century involved studying people who'd had the two halves of their brain severed as a radical treatment for epilepsy. This had some bizarre consequences, including patients sometimes performing contradictory actions – such as one hand doing up buttons while the other undid them. Yet throughout, the patients retained a coherent sense of self. They even invented reasons to explain away their bizarre behaviour – their brains were literally making up explanations on the fly to preserve the narrative of a unified self.

> **"OUR SENSE OF SELF ALSO EXTENDS TO THE STUFF WE OWN"**

In healthy people, psychology studies have revealed the quirks of memory that help to reinforce this constructed sense of self. For instance, we're more likely to recall and rehearse memories that are consistent with how we see ourselves. Imagine you think of yourself as an introvert – it's likely you'll find it easier to access and describe

AMY ARTHUR
Health & Wellness



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Health

memories from your past that support this idea of the kind of person you are. You're effectively editing your autobiography to fit your current self-concept.

A key region orchestrating this construction is the medial prefrontal cortex, which sits right at the front of your brain, just behind your forehead. In studies, when researchers ask people to identify which traits best describe them, either now or in the future, it's this part of the brain that flickers into action, far more than when making similar judgments about other people.

Our constructed sense of self also extends to the stuff we own. When people are put in a brain scanner and asked to view a series of objects, the medial prefrontal cortex tends to light up when they see their possessions. But not when they're shown a random object that doesn't belong to them. This shows how quickly and flexibly our brains redraw the boundaries of self.

Memory processes are also crucial to this ongoing construction. Damage to the hippocampus (located deep in the brain, in line with the temples) can stop people from being able to imagine themselves in the past – or in the future – revealing how dependent our sense of continuous identity is on active brain processes.

Your brain doesn't just construct your sense of self through time, it also maintains it in space, so that you have a stable feeling of ownership of your body. A different part of the brain, known as the temporo-parietal junction (located behind your ears), plays an important role in this aspect of your identity. In a study from 2005, when researchers electrically stimulated this brain region during brain surgery, it triggered an out-of-body experience in the patients. They felt as if they were floating outside of themselves.

In short, our sense of a stable self usually feels utterly convincing, yet it can be fractured by brain damage and clever neurological experiments. Taken together, the evidence suggests your sense of me-ness is a construction, one that your brain works tirelessly to maintain. ***CJ***

INKFISH LLC, ALAMY ILLUSTRATION: ROBIN BOYDEN

FRANCES BROWNING, VIA EMAIL

WHAT'S THE DEEPEST A SUBMARINE HAS GONE?

All the way to the bottom! The deepest part of the ocean is the Mariana Trench, the narrow pinch point where the Pacific tectonic plate churns its way back into the mantle beneath. And the deepest point along that line is the Challenger Deep – over 10,900m (35,700ft) deep.

If you turned Mt Everest upside down, so that it extended down from sea level, its peak would still be over 2km (6,500ft) from the Challenger Deep's bottom.

The first submarine to reach the bottom was the *Trieste* in 1960, piloted by Don Walsh and Jacques Piccard. Their depth record has since been pipped by a few metres, by explorer Victor Vescovo. His submarine *Limiting Factor* (right) made numerous trips to the bottom and recorded a depth of 10,928m in 2019. ***LV***

NIKKI COSTA, VIA EMAIL

HOW DO HULA HOOPS STAY UP?

Hula hoops rotate by rolling around your body, like a wheel on the road, except that they roll on their inside edge instead of the outside. If you gyrate your hips in sync with this rotation, your waist will push against the hoop at just the right point to apply a force that keeps the hoop spinning.

The hoop stays up because your hips are wider than your waist, so pushing sideways also causes the hoop to slide up this slope to the narrowest point.

A 2024 study at New York University confirmed that an hourglass waist was the best figure for hula hooping. ***LV***

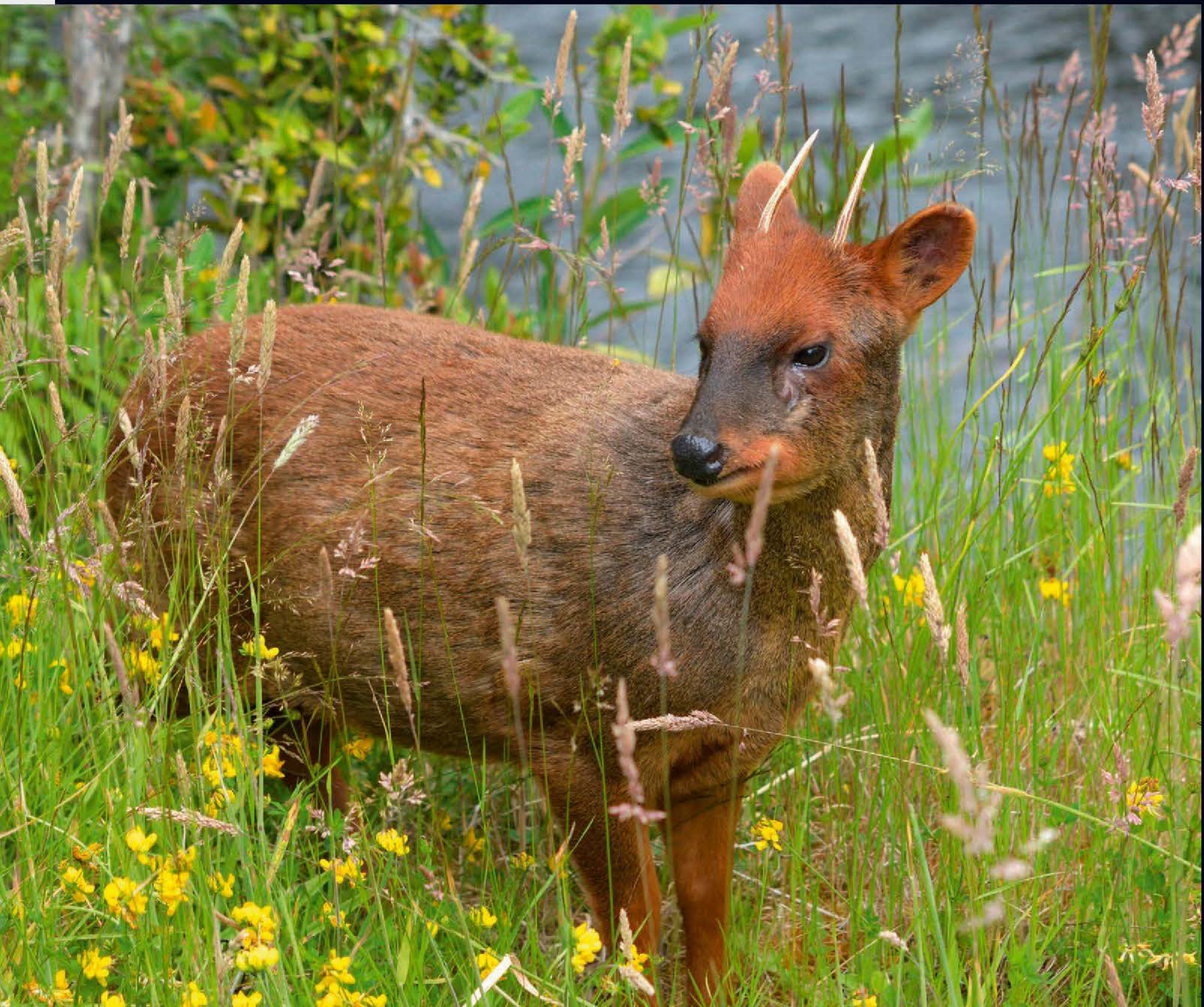
THE PUDU

> "THESE ARE NERVOUS ANIMALS THAT MOVE CAUTIOUSLY AND SPOOK EASILY"

Just when you thought Bambi couldn't get any cuter, meet the pudu, the world's smallest deer. Standing little taller than a domestic cat, what it lacks in size, it more than makes up for in allure. Doe-eyed, button-nosed, with little legs and perky ears, this diminutive South American mammal looks like it has stepped straight out of a Disney film.

There are two species of pudu: southern and northern, aka small and smaller. The southern pudu, which has chestnut-brown fur, is native to the Valdivian temperate forests of south-central Chile and Argentina. Meanwhile, the northern pudu, which has a lighter coat and a darker face, is native to the Andes of Colombia, Venezuela, Peru and Ecuador.

Adult northern pudus weigh about the same as a domestic moggy, but it's here that the cat similarities stop. Pet cats don't willingly sport fancy headgear, but pudus do. Every year, adult males grow a pair of smart, single-pointed, backwards-facing antlers that they use for 'jousting.' In the rutting season, which falls in the southern hemisphere autumn, males compete with each other to cement their territory and establish dominance and mating rights.



Fights get messy. Males jump, kick, thrash with their forelegs, chase one another and lock antlers. Think Bambi meets bare-knuckle brawling.

The rest of the time, however, they keep themselves to themselves. If they're not males fighting, or females raising their fawns, pudus live a solitary existence. In the wild, they're hard to spot. During the day, they lay low in the dense forest undergrowth, but as the light fades, they sneak out to do important pudu jobs, like marking their territory with dung, and eating.

When it comes to food, pudus favour the low-hanging fruit, metaphorically and literally. Because of their diminutive stature, they readily eat plant material that can be found on or near to the ground, such as herbs, ferns, tree bark and fallen fruit. If they fancy a snack from higher up, however, they'll either stand on their hind legs or climb branches to reach it. And if that doesn't work, pudus have been observed using their front legs to press down on saplings until they bend or snap, all for the juicy, fresh leaves.

These are nervous animals that move cautiously and spook easily. This is understandable, because when you're little, everything seems big, including the Andean foxes, horned owls and cougars, which eat pudus. When frightened, pudus bark an alarm call, before zigzagging pronto to the safety of the undergrowth.

Although they breed well in captivity, in the wild, things aren't so peachy. Pudus are in danger from the destruction of their forest habitat, which is being cleared for cattle ranching, agriculture and logging. They're also captured for the pet trade and killed for food by specially trained hunting dogs. A recent study has also found that wild pudu are picking up infections from neighbouring farm animals and while the consequences of this remain uncertain, one thing is for sure: this is a little deer that deserves a lot more respect. ***HP***

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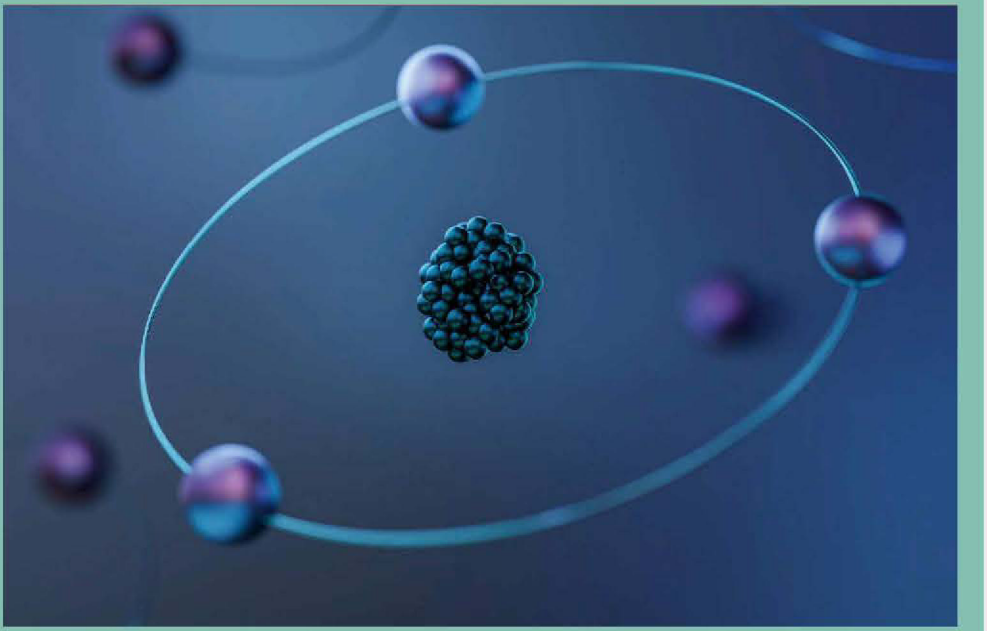


ALISSA SCHNEIDER, LONDON

HOW MANY ELECTRONS ARE THERE IN THE UNIVERSE?

It's difficult to know for sure how many electrons are in the Universe. British physicist Arthur Eddington (1882–1944) came up with a number in 1938, now known as the 'Eddington number'. Eddington's calculation was actually for the number of *protons* in the Universe, but based on our knowledge of the physics of the early Universe, and the known strength of the electromagnetic force, we can assume this is the same as the number of electrons. Eddington's number was 1.57×10^{79}.

Unfortunately, this estimate was based on rather dubious physics. More modern estimates put this number slightly higher, at almost 10^{80} (that's 1 followed by 80 zeros).

However, according to American theoretical physicist John Wheeler (1911–2008) there could be only *one* electron in the Universe. The story behind this idea was first recounted by physicist Richard Feynman (1918–1988) during a speech for his jointly awarded 1965 Nobel Prize in Physics. Feynman said that Wheeler telephoned him in the spring of 1940, saying, "I know why all electrons have the same charge and the same mass". When asked to explain, Wheeler said, "because they're all the same electron!"

Wheeler had realised that, mathematically speaking, electrons and their antiparticles, positrons, could be thought of as the same particle, but moving in opposite directions through time. Electrons have negative charge and move forwards through time, while positrons have positive charge and move backwards.

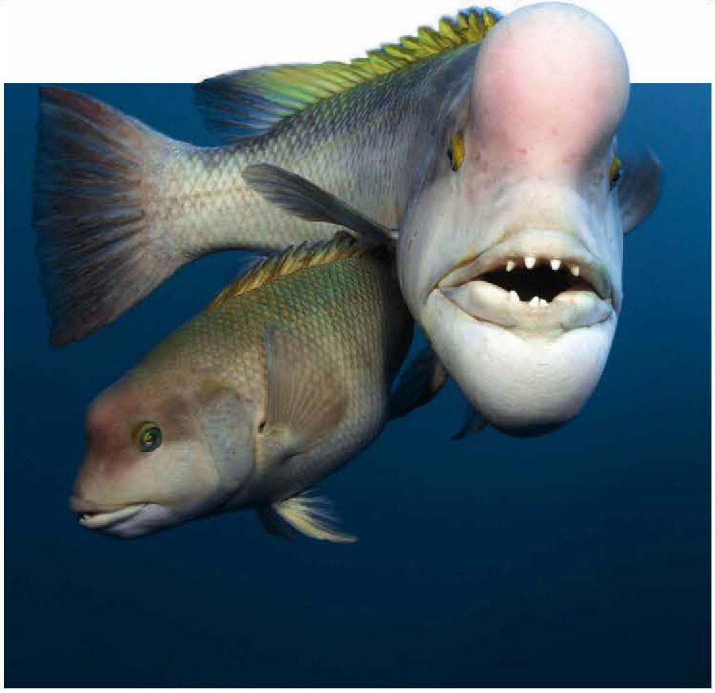
But, thought Wheeler, particles could move through 'space-time' in complex ways, so a single particle could trace out a tight 'knot' of countless trajectories in both space and time. We could see this particle manifest itself at multiple locations simultaneously, giving the impression that it's different particles. The electrons we see would correspond to the trajectories moving forward through time, and the positrons to trajectories moving backwards through time.

There are problems with Wheeler's idea. The most important is that the Universe contains many more electrons than positrons (and there's more matter than antimatter), while the one-electron theory suggests these should be equal. Wheeler originally suggested, unconvincingly, that the missing positrons could be 'hiding' somewhere, perhaps inside protons. The one-electron theory may also violate the principle of causality.

This single-electron theory was never pursued by Wheeler, or anyone else. It's now regarded as an interesting, but discounted, 'thought experiment'. However, the idea of the time-reversal of electrons and positrons was a major influence on some of Feynman's later work. ***AG***

ALEX JACKSON, VIA EMAIL

HOW CAN ANIMALS SWITCH SEX?



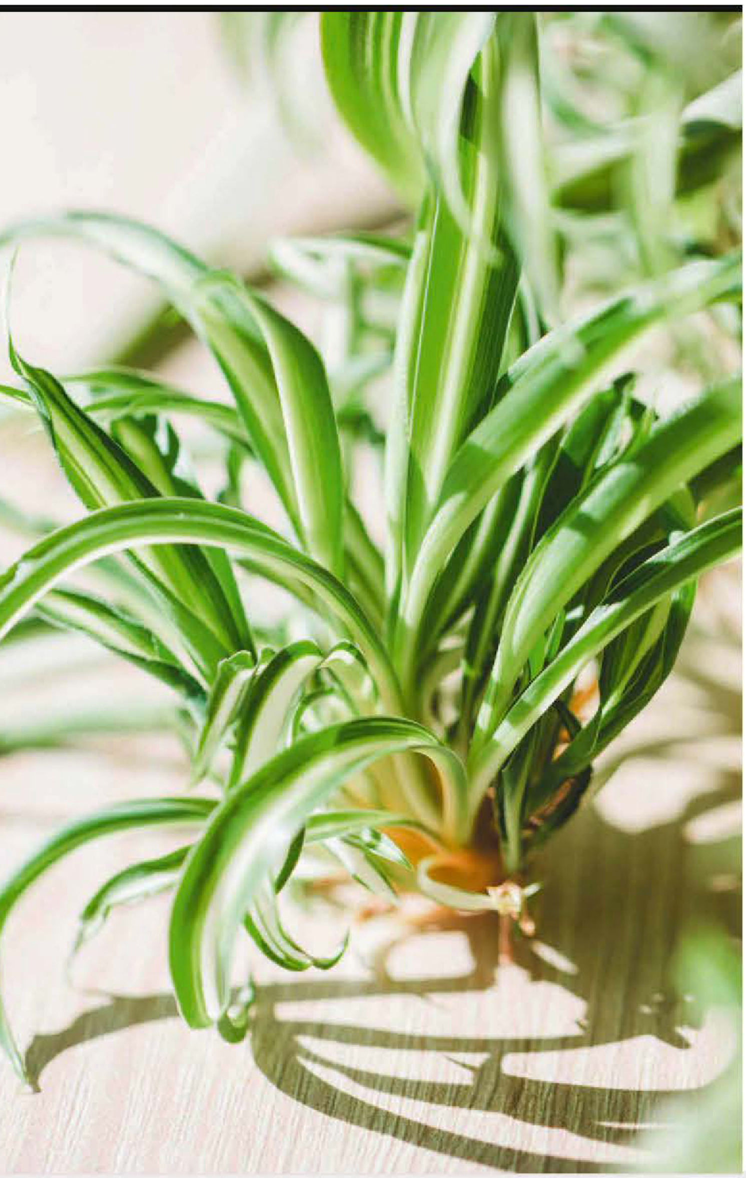
About two per cent of the world's fish species – some 500 species – are thought to switch sex at some point in their adult lives. Some, like the Asian sheephead wrasse (right), routinely change from female to male. Others, like the clownfish, change from male to female, while some, such as coral-dwelling gobies, switch back and forth depending on what's happening in the environment.

This is possible because, unlike mammals and birds, the sex of many fish species isn't determined by the presence of sex chromosomes. Instead, environmental cues trigger changes in gene activity, which then change levels of key enzymes and hormones. In particular, one enzyme, called aromatase, is known to change male sex hormones into female sex hormones, which can then transform gonads into ovaries.

Sometimes, the environmental cue can be social. Clark's anemonefish, for example, is a type of clownfish that lives inside protective anemones. The fish live in small groups, with a breeding

EUGENE ABBOTT, NORTHUMBERLAND

WHICH HOUSEPLANT IS BEST FOR AIR QUALITY?



Plants remove carbon dioxide from the air through the process of photosynthesis and release oxygen as a byproduct. They also help clean the air of pollutants, but it's not clear which houseplant is the best at it.

In 1989, NASA researchers tried to find out if plants could clean the air in sealed environments, like the ones aboard a space station. Different houseplants were put inside small sealed chambers with carbon monoxide, benzene and formaldehyde (all common pollutants found in the air inside a typical home).

Based on NASA's tests, spider plants were effective at reducing carbon monoxide concentrations from 130 parts per million (ppm) – well over the generally accepted maximum safe indoor level of 9ppm – down to zero over the course of 24 hours. *Philodendron domesticum* (aka spade leaf) was the best at clearing high concentrations of formaldehyde and *Gerbera jamesonii* (aka Gerbera daisy) did the best job of clearing benzene.

It's important to note, however, that the plants were tested in chambers much smaller than the typical room, and the levels of formaldehyde and benzene remaining after 24 hours, were still higher than the generally accepted maximum safe concentrations. Also, our homes aren't sealed environments. The air exchange through windows and doors affects plants' phytoremediation abilities, making them less efficient at cleaning their surroundings.

Studies that use real-world conditions do show small improvements in air quality thanks to houseplants, but not on the scale of the NASA results. ***AA*** male and female pair, and a number of non-breeding subordinates. If the breeding female dies, however, the largest subordinate male will switch sex and take her place.

Other times, the cues can be direct changes to the water. It has been shown that when certain pollutants are flushed into rivers, they cause male fish to develop female characteristics, such as egg production. Meanwhile, a 2008 study found that an increase in water temperature of just 1–2°C can skew the sex ratio of some fish towards more males.

Some of these transitions are helpful. When clownfish switch sex, it's because evolution has selected this as a useful strategy that helps the fish to keep breeding and the species to survive. Not so, sex changes that are driven by human activity. When we dump chemicals into rivers or cause the oceans to warm, we play a dangerous game with the futures of all the species that live there. ***HP***

ZACK EVERETT, MANCHESTER

WHAT'S THE LONGEST-LIVING INSECT?

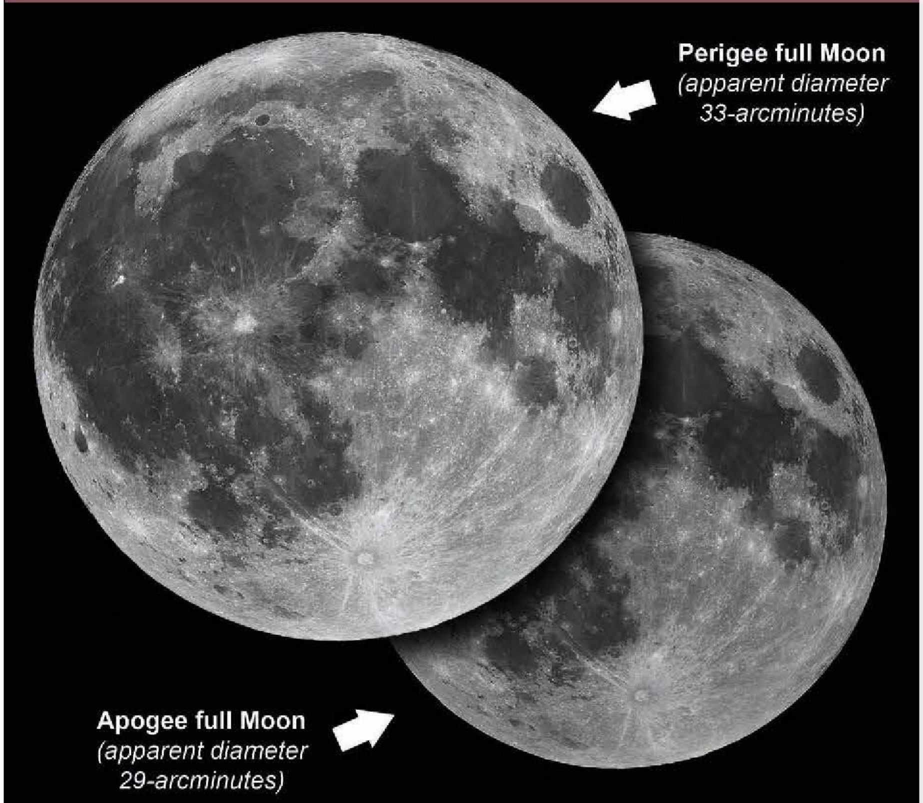
We often think of insects as being short-lived, so you might be surprised to learn that some insects can live for decades!

The record for the longest-lived insect goes to the golden jewel beetle (*Buprestis aurulenta*), which can spend as long as 47 years in its larval form, although the adults only live a few months.

But the insects with the longest documented adult lifespans are ants and termites. Queens of the black garden ant (*Lasius niger*) have been reported living 28 years in laboratory colonies, and foraging termite (*Macrotermes*) queens are thought to live up to 20 years. ***CA***

NATURE PICTURE LIBRARY, ALAMY ILLUSTRATION: PETE LAWRENCE

ASTRONOMY FOR BEGINNERS



HOW TO SEE THE FULL MOON AND GEMINIDS

WHEN: EARLY DECEMBER

The upcoming full Moon on 4 December is astronomically known as a perigee full Moon. Perigee is a term that describes when the Moon is closest to Earth in its elliptical orbit. Consequently, perigee full Moons are slightly larger and brighter than average. To be honest, from month to month you wouldn't really notice the difference. This is because the transition from an apogee full Moon – one that occurs when the Moon is furthest from Earth in its orbit – and a perigee full Moon, is a gradual affair throughout the year.

This year's December full Moon may make more of a stir because it occurs when the Moon is in Taurus, where it'll be able to perform a high arc across the sky. When at its highest point in the sky, due south, the Moon will appear approximately two-thirds up the sky. Compare this with the full Moons that occurred in the middle of the year when it was in Sagittarius. These barely made it 10° above the horizon (or the width of a clenched fist at arm's length) when highest. Big and bold in the early December sky, if it's clear, the December perigee full Moon will make a bold, bright statement.

A downside of this, is that it'll be hard to see much else besides the Moon. But don't despair! It means that by the middle of December, the Moon will be largely out of the way for the best meteor shower of the year: the annual Geminid shower. This reaches its peak on the night of 13/14 December with a zenithal hourly rate (ZHR) of 150 meteors per hour. This is a theoretical rate assuming perfect viewing conditions, the visual hourly rate, or what you're likely to actually see, will be lower but still respectable, especially into the early hours of 14 December. ***PL***

by PETE LAWRENCE
(@Avertedvision)
Pete is an astronomy expert and presenter on The Sky at Night.

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BBC FOUR

HUEY ROBBINS, VIA EMAIL

WHICH VAPE FLAVOUR IS WORSE FOR YOU?

If you're trying to quit smoking, you'll have probably heard talk that switching to e-cigarettes – or vapes – is a healthier option. One study by researchers at University College London estimated that in 2017 alone, over 50,000 people stopped smoking thanks to their use of e-cigarettes.

It's true that most of the compounds that cause smoking-related diseases aren't found in e-cigarettes (which, unlike heated tobacco products, don't burn tobacco), so vaping is safer in that sense. But if you've never been a smoker, it's not advisable to take up vaping.

The e-liquid that's used in vapes typically contains a mix of propylene glycol and glycerine. Both compounds are considered safe to ingest and though they haven't been officially labelled safe for inhalation, a 2020 review of clinical studies found "little impact on lung functions, as well as no significant inflammatory effects."

Added to this mix are various synthetic flavourings, which, in order to comply with the regulations of the country they're being sold in, must be considered safe for use as ingredients in foodstuffs (though not necessarily for inhalation). Many e-cigarettes also contain nicotine, which is helpful for those trying to quit smoking without suffering withdrawal symptoms, but could be addictive for new users and has been linked to lung cancer in mice.

Anyone who's ever peeked into a vape shop will know there are hundreds, if not thousands, of flavours available. There are the 'traditional' flavours of tobacco and menthol, but also fruit and citrus flavours, and even e-liquids that mimic baked goods.

Studies have suggested that fruit flavours may be the worst vape flavours for your health. Research on cell samples from 2016 associated strawberry flavour with higher cell toxicity, meaning it could cause more cellular damage. More recently, a 2019 study linked fruit flavours to the presence of two compounds, acrylonitrile and acrolein, which are known to have carcinogenic potential and can cause lung irritation.

One study published in 2024 found that inhalation of berry-flavoured e-cigarette vapour effectively paralysed the immune cells in the lungs of mice. When these mice were then exposed to harmful bacteria, their cells struggled to clear the infection. Compared with healthy controls, the vape-exposed mice had a lower survival rate and those that did survive took longer to recover from their infection.

This doesn't mean the non-fruit flavours are 100-per-cent safe, however. The chemicals in e-liquids can be unstable, so they can react to form new compounds when they're heated by the e-cigarette to form the breathable vapour. One group, the aldehydes, are commonly used as flavourings in vanilla, cherry and cinnamon vapes, but chemical reactions within the e-cigarette can create acetals, which are known respiratory irritants. There may be other chemical reactions occurring between other flavours that we haven't yet identified: this area of research is still very young, especially compared to the wealth of knowledge available about smoking and its effects on people's health.

So, while e-cigarettes are often recommended to those who are trying to quit smoking, there are risks involved with taking up vaping. If we know anything, it's that we still don't completely understand what vaping does to the body. ***AA***

> **"RESEARCH ON CELL SAMPLES ASSOCIATED STRAWBERRY FLAVOUR WITH HIGHER CELL TOXICITY"**

JEANETTE SULLIVAN, VIA EMAIL

DOES NEGATIVE THINKING INCREASE YOUR RISK OF DEMENTIA?

Dementia and disorders like depression and anxiety (conditions associated with persistent negative emotional thoughts) have long been intertwined. Indeed, distinct screening tools have been developed to differentiate between depression and dementia (because "being cognitively incapable of answering questions" and "being insufficiently motivated to answer" look the same on a test).

It makes sense, in a way. Whether it's the knowledge or the symptoms of a dementia diagnosis, 'prolonged low mood' is inevitable. However, a recent study suggests that constant, repetitive patterns of negative thinking, a 'fatalistic attitude', could lead to earlier onset or amplified symptoms of dementia. In short, constant negative thinking could cause or amplify dementia.

How could a negative mindset lead to actual physical harm to the brain? With the caveat that it's by no means confirmed that it does (and even if it were, there are many complex factors at work), one possible mechanism is via the stress response.

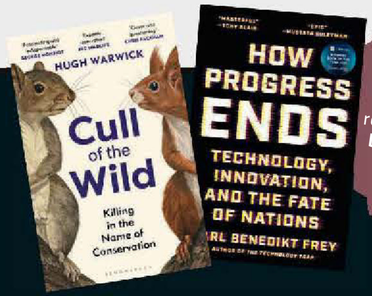
A typically functioning human brain produces an optimism bias in our thinking. We tend to assume we're right about stuff, that fairness is paramount and that things will go our way. This keeps us confident and motivated, and prevents us from worrying about anything and everything. It's a psychological defence mechanism, of a sort.

But when we lose this optimism, as often happens in depression, we experience far more stress and negativity. And the chemical element of stress is known to cause biological stress to the brain, often resulting in the more familiar mental disorders.

If your brain was already physically compromised by whatever factors cause dementia, then it wouldn't be far-fetched to say that constant negative thinking could speed up the whole process.

It's usually trite to say, "Try not to think about it", but here it may well be medically sound advice. ***DB***

GETTY IMAGES, ALAMY ILLUSTRATION: ROBIN BOYDEN


HUGH WARWICK
Cull of the Wild
Killing in the Name of Conservation
HOW PROGRESS ENDS
TECHNOLOGY, INNOVATION, AND THE FATE OF NATIONS
RL BENEDIKT FREY

WRITE IN
The writer of next issue's Question of the Month will receive **two popular science books**: *Cull of the Wild* and *How Progress Ends* **worth over £35!**

TERENCE COLDWELL, YORKSHIRE

CAN YOU CATCH COVID ON TOP OF COVID?

As a doctor, I often hear from patients worried about COVID-19, especially those with long COVID. One question comes up a lot: 'Can you catch COVID again while still dealing with long COVID, maybe a different strain?' Many people assume that having long COVID means they're still 'immune' to getting the virus again. But sadly, this isn't the case.

Long COVID results in symptoms like tiredness, trouble breathing or brain fog that sticks around for months after a COVID infection. Unfortunately, having long COVID doesn't protect you from getting the virus again.

Your body's protection from a past infection or the vaccine fades over a few months. Plus, new versions of the virus, called variants (like Omicron's KP.3 or XEC in 2025), can slip past your immune system. This means that even if you're still managing long COVID symptoms, you can catch COVID again and, in some cases, it might trigger a flare-up or slow down your recovery.

A positive test could mean the same or a new variant, but either way, it's still COVID. Vaccines, especially the 2025 boosters, help lower the risk of serious illness. If you have long COVID and test positive, rest, drink plenty of water and talk to your doctor if your symptoms get bad.

COVID is still with us and it continues to evolve. So, it's worth continuing some of those protective habits, like testing if you feel unwell, wearing a mask in busy indoor spaces and keeping up with vaccinations. These small steps can help reduce your risk and protect those around you, especially if you're still navigating the long tail of this virus. ***NM***

QUESTION OF THE MONTH

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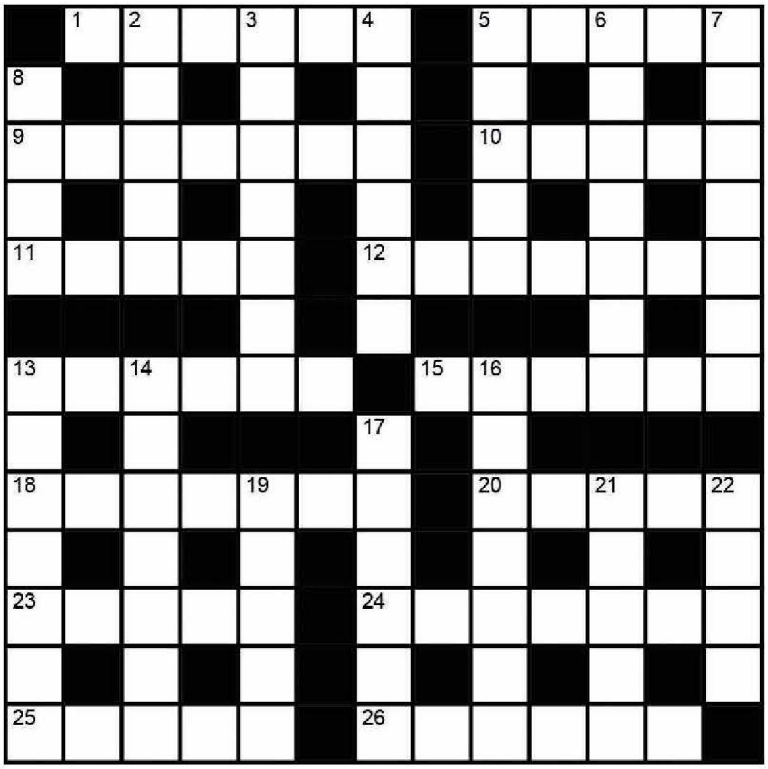
CROSSWORD

PENCILS AT THE READY!



ACROSS

- **1** Agent has even less money (6)
- **5** New room, new idiot (5)
- **9** Calm about finding articles in calendar (7)
- **10** Playground item shows change of opinion (5)
- **11** Alter design at some future time (5)
- **12** Nobleman and former queen holding one in the past (7)
- **13** Way to travel shows pace (6)
- **15** Set out a site behind loud carnival (6)
- **18** Firm trade negotiated before movement (3,4)
- **20** Hammer donated to head of library (5)
- **23** Sit, holding last of writer's writing (5)
- **24** Gull, say, cooked - braised (7)
- **25** Survives final son (5)
- **26** He sets out arguments (6)

DOWN

- **2** Right time to come back and send money (5)
- **3** Related to considerate communist (7)
- **4** Dodgy business involving sports equipment (6)
- **5** Commiserated about skinflint (5)
- **6** Almost bringing up singular fruit (7)
- **7** Airing problem about European country (7)
- **8** Everything after wife is an obstacle (4)
- **13** Small demand around edges of palace for a knife (7)
- **14** Oar isn't transported in provisions (7)
- **16** Tearing off to see an ungracious person (7)
- **17** Pamper island group (6)
- **19** Drains top off pitchers (5)
- **21** Bad habit, including old opinion (5)
- **22** The game I play in Latin (4)

ANSWERS For the answers, visit **bit.ly/BBCFocusCW**
Please be aware the website address is case-sensitive.

GETTY IMAGES

NEXT ISSUE

UNKNOWN HORIZON

Some experiences at life's end hint at mysteries that extend beyond understanding

PLUS

COLD COMFORT

Science-backed habits to help you dodge this season's sneezes and sore throats

UNIVERSAL ODDS

Are we inevitable, designed, or an improbable accident in an infinite multiverse?

ON SALE 9 DEC

SF

BETTER LIVING THROUGH SCIENCE

PASS THE PLASTIC

All of us are ingesting microplastics. Could dietary fibre help us get it out?

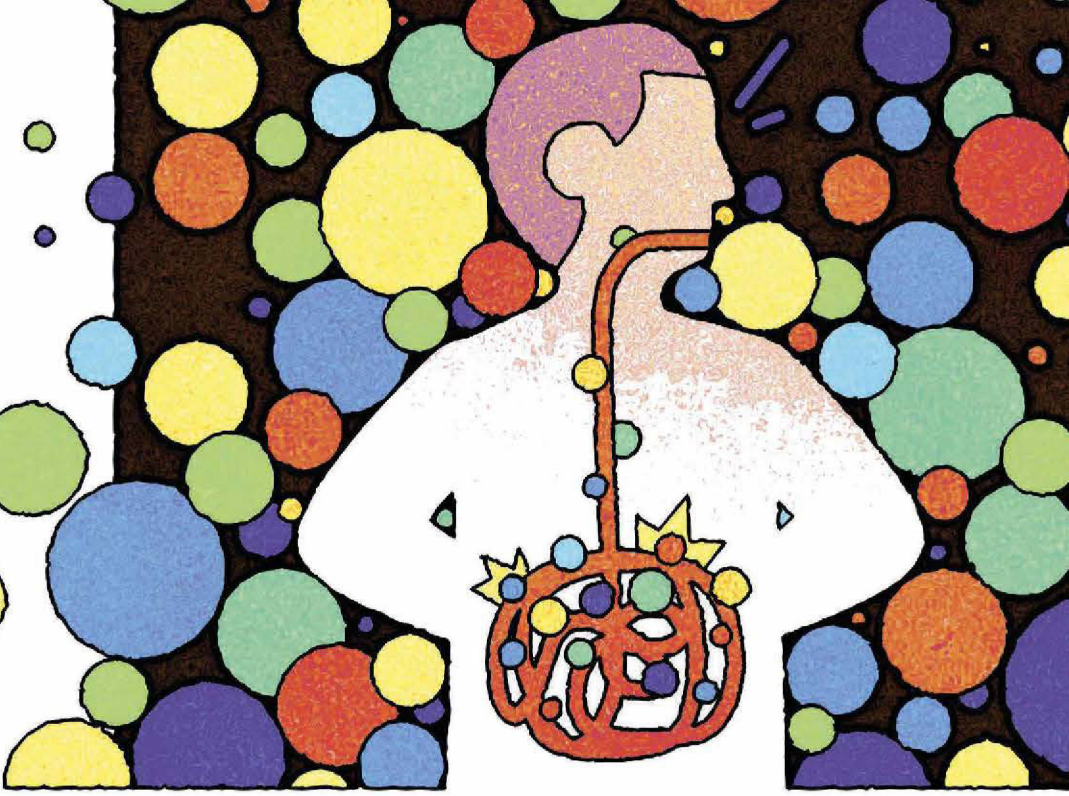


ILLUSTRATION: KYLE SMART

by **IAN TAYLOR**
Ian is a freelance science writer and the former deputy editor of BBC Science Focus.

It's a difficult truth to swallow, but research shows that, every year, we consume between 39,000 and 52,000 microplastic particles with our food. It's enough to give you a kind of cognitive indigestion. How, in this age of food safety and nutrition, can we be pigging out on plastics when unappetising studies show they're probably harming our short- and long-term health?

One 2024 study looked at the plastics found in a range of 16 sources of protein in US diets. It found that from those foods alone, the average meal contained between 74–220 microplastic particles. That's not counting the flecks that come from our drinks bottles or food containers. It doesn't account for the plastics that break off our cookware and end up on our plates, either.

Microplastics have also been found in drinking water, salt, rice, honey and powdered supplements. They leech from teabags and chip off plastic chopping boards. Even fruit and vegetables contain microplastics absorbed through their roots from contaminated soil and water.

Plastic is everywhere in our food system and research is only beginning to give us a clear idea of how it affects us. Studies have shown links between microplastic exposure and increased risk of cardiovascular disease, some cancers and metabolic conditions. As well as physically damaging our tissues, they can cause inflammation, cause changes to our microbiome and expose us to harmful chemicals such as per- and polyfluoroalkyl substances (PFAS), phthalates and bisphenol A.

There's a morsel of optimism, however. Researchers are beginning to wonder if dietary fibre can lower the amount of microplastics accumulating in our digestive systems. A 2024 paper speculates that fibre's absorbent properties may allow it to bind to microplastics in the gut, allowing us to secrete them in greater quantities – plastic poo in your toilet bowl, in other words.

The idea is that soluble and insoluble fibres form a gel-like barrier that stops microplastics from crossing the intestinal wall and entering the bloodstream. With nowhere else to go, plastics are swept along with other waste.

This mechanism has not been studied or directly observed in humans as yet, but earlier this year, a research team in Japan did show something similar working in rats. Scientists at Tokai University fed rats a selection of indigestible dietary fibres mixed with microplastics. They found that rats fed with a type of fibre called chitosan excreted significantly more microplastics than others.

"We confirmed that chitosan binds to microplastics," says study author Prof Muneshige Shimizu. "We believe it could be utilised in any food where the chitosan structure can be maintained."

Shimizu says that not every type of fibre they tested did the same thing, so further research is needed to figure out which specific structures are effective.

In the meantime, there may be other ways that fibre reduces the health toll from microplastics. Researchers at Boston University in the US found that in mice, a fibre supplement could flush out PFAS, the so-called forever chemicals that plastics are often treated with. Researchers found that gel-forming fibres act as a kind of magnet for PFAS in the gut, again helping to direct them out of the body.

But before you stockpile a year's worth of Weetabix, it's important to note that researchers are still working to understand which kinds of dietary fibres help to flush out plastics and PFAS. Nevertheless, increasing your fibre intake is generally recommended for a wide range of health reasons, from cardiovascular health to cancer risk reduction.

Although microplastics are unavoidable in the modern world, there are other ways to lower the amount you're exposed to in the kitchen. Dr Lisa Zimmermann is from the Food Packaging Forum, a non-profit foundation that researches how chemicals from packaging affect what we eat. She tries to minimise the plastics she's exposed to by getting fruit and vegetables from farmers' markets and avoiding things like disposable cups lined with plastic.

She also avoids microwaving plastic containers. "The heat leads to faster particle movement and particle release," she says. "I use glass or ceramic instead.

"You can't completely avoid plastics, but I try to at least reduce them." **SF**

"PLASTIC IS EVERYWHERE IN OUR FOOD SYSTEM"

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